INFLAMMATIONS OF THE LIVER

AND THEIR SEQUELÆ

DR. GEORGE HARLEY, F.R.S.

BY THE SAME AUTHOR.

In clear type, and with a full Index.

DISEASES OF THE LIVER.

WITH AND WITHOUT JAUNDICE.

"Twenty years ago, Dr. George Harley published a monograph on Jaundice . . . . which made him widely known as a highly accomplished physician and an able and original student and interpreter of the etiology, diagnosis, and treatment of the diseases upon which he wrote. He has now given the profession the fruits of his twenty years further, and greatly enlarged, experience of the diagnosis and management of Diseases of the Liver. . . . . The number and variety of the subjects dealt with are marvellously great, and the treatment of each one shows some originality of views and handling."—*Medical Times.*

"This is one of the freshest, most readable, and most instructive medical works that have been laid on our table during the present decade."—*Philadel. Med. Times.*

"For originality of thought and expression, and diagnostic sagacity . . . this work stands unexampled. It is undoubtedly the most able work on the subject, and deserves to rank as a classic. . . . . Charmingly written in sparkling eloquent style. . . . . The remarks on germicides are deeply interesting and beautifully penned. The chapters on Biliary Concretions and Gall-Stones are very fully wrought out. This book may be justly regarded as the most original effort of the times."—*The Aust. Med. Gaz.*

"The Medical Profession, both in England and America, has for some time been on the *qui vive* for this new work on the Liver, both because hepatic literature is meagre in the extreme, and because it was well-known that Professor Harley was specially qualified to write an authoritative work on the subject. . . . . The author has unquestionably written the most valuable work on Hepatic Disease that ha yet appeared. . . We must confess that we have tried, and tried hard, to find some error in the work, to preclude the charge of partiality being made."—*Virginia Med. Monthly.*

"This is an entirely new book, and the most complete which exists *in a practical point of view.* We know of none other which it is possible for us to compare advantageously with it . . . for it is quite a remarkable book . . showing, as it does, the importance of physiology and chemistry in the diagnosis of liver disease . . . and is distinguished by the originality of the views, as well as its eminently clinical character."—*Journal de Med. de Paris.*

"It is a storehouse of practical information, and a standard treatise *sui generis.*"—*Canad. Pract.*

"This work is a most excellent and thoroughly practical one. It is largely a clinical work, containing a number of instructive cases, and is written with a most enviable freshness and vigour. . . . . As a guide to practice in making a diagnosis, and formulating a rational prognosis and treatment, it will compare favourably with any work on the same subject, which has ever appeared—a strong statement, no doubt, but one which will be fully endorsed by anyone who has had the pleasure of reading the book."—*Edin. Med. Jour.*

"It is, indeed, refreshing to find such men as Dr. George Harley engaged in book making. . . . He presents us a work of rare practical value on Diseases of the Liver. . . . The whole subject matter is treated in such a masterly manner that the work is destined to find a place among the classics."—*The Medical Herald, Louisville.*

"The known ability of the author, and the fact that he has for many years made a special study of the physiological chemistry of the excretions renders his present work a valuable addition to the literature of the subject."—*Canada Med. and Surg. Journal.*

"The Medical Profession in America may congratulate themselves that this book has been reprinted in this country. The author may be congratulated on having written a standard work on the Liver. It is a book of really very great merit, and undoubtedly the best authority on the liver that can be found. While it is an eminently scientific book, it is at the same time a very practical one. . . . The book is a credit to both author and publisher, and an invaluable addition to medical literature."—*Med. Regist.*

"This work is the most complete work upon Diseases of the Liver now before the profession. It embodies not only the results of his own large experience and observations; but also exhibits the researches of others. As a scientific treatise on hepatic affections and their treatment it will certainly hold a first position among standard works."—*Cincinnati Med. News.*

"The reader, having taken the volume up, does not care to lay it down; and there is no one who will not have his knowledge improved by perusing it. It is beautifully got up, has many fine illustrations including chromolithographs, and an exhaustive Index."—*The Medical Press.*

1186 pages and 38 Illustrations. Price One Guinea.

Also

SOUNDING FOR GALL-STONES;

Containing a *résumé* of their most Salient Signs and Symptoms; together with the Difficulties and Dangers Encountered in their Symptomatic Diagnosis. Price Sixpence.

J. & A. CHURCHILL, 11 NEW BURLINGTON STREET, LONDON, W.

INFLAMMATIONS OF THE LIVER,

AND THEIR SEQUELÆ.

INFLAMMATIONS OF THE LIVER,

AND THEIR SEQUELÆ:

ATROPHY,

CIRRHOSIS, ASCITES, HÆMORRHAGES, APOPLEXY,

AND

HEPATIC ABSCESSES.

BY

DR. GEORGE HARLEY, F.R.S.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS; CORRESPONDING MEMBER OF THE ACADEMY OF SCIENCES OF BAVARIA, OF THE ACADEMY OF MEDICINE OF MADRID, AND OF SEVERAL CONTINENTAL MEDICAL SOCIETIES; FORMERLY PRESIDENT OF THE PARISIAN MEDICAL SOCIETY; PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL; AND PROFESSOR IN UNIVERSITY COLLEGE, LONDON.

LONDON:
J. & A. CHURCHILL,
11 NEW BURLINGTON STREET.

1886.

PREFACE.

ALTHOUGH no one can doubt that the age in which we live amply belies the validity of the dogma that "There is nothing new under the sun." From the transparent fact that while in the field of science discovery follows upon discovery; in that of art invention treads upon the heels of invention, and in everything else around us daily and hourly improvement succeeds improvement. There are, nevertheless, still to be met with persons who fail to perceive that Medicine is no exception to the rule, and that in her sphere too "old things have become new." Even within the pages of this little book, notwithstanding that it is limited to the consideration of matters connected with one department of medicine alone, those versed in the literature of the subjects of which it treats will find evidence ofthe justness of the above remark.

It will be noted, that I specially say those versed in the subject, I am moved to do this from it being an irrefutable fact that, precisely as it requires one to

be possessed of a certain amount of talent in order to enable him to appreciate talent in another, so in like manner it equally requires a certain amount of knowledge of any given department of medicine to enable a reader to guage with exactitude either the extent or the value of the improvements a treatise upon it portrays. In spite of this necessity, however, it is not improbable that even those having only a limited acquaintance with modern hepatic literature will perceive that the essays composing this volume are neither a compilation of other men's labours nor a *rechauffé* of other people's thoughts, nor even resemble a text-book for beginners, in being copiously padded with appropriate quotations, or made to look learned by the introduction of numerous references to the writings of others. Indeed, from these essays having been penned for the use of those already familiar with the ordinary accepted facts and theories connected with the matters handled, they mainly consist of new data, and consequently may be said to be original in the ordinary sense of the word. No human ideas are absolutely *ab initio* original. From the fact that the germ of all, so-called, original thought being of necessity the product of personal observation and generalisation, supplemented by ideas derived from hearing and reading the opinions emanating from other intellects.

The only pretence these essays have to be considered original, then, is in so far as they embody the views and experience gained by their writer during a

quarter of a century's work in the special department of medicine of which they treat. Together with the digested and assimilated ideas he has culled from the writings and conversations of others.

The chapters on hepatitis and abscesses, including their causation, diagnosis, and treatment, were published in the *Medical Press and Circular* during the months of January and February, and the first week of March of this year. Whereas, those on atrophy, cirrhosis, ascites, and hæmorrhages, now appear for the first time. Thus freighted the *livraison* is launched on the ever changing ocean of medical credence. Where, although perchance, like many other books of a similar character, from its containing a series of practical as well as theoretical innovations, it may be destined for a time to be tossed amid the stormy billows of controversy, and not improbably, even, have to run the gauntlet of the ire of ignorance. From the writer being neither in the habit of promulgating doctrines before they have undergone careful consideration, nor putting forth ideas he does not himself believe in, he is sanguine enough to imagine that those herein embodied will not alone be able to hold their ground, but will, in due course, safely reach the calm haven of acceptance.

25 Harley Street, London, W.

CONTENTS.

PAGE

Improvements that have recently taken place in the Diagnosis and Treatment of Liver Diseases... ... 5

Novelties in Gall-stone Formations 9

Extrusion of Biliary, from Gall-Bladder, and Urinary Concretions from Ureters by Digital Manipulation... 10

Difficulties in the way of Diagnosing Inflammations and Abscesses of the Liver... 11

Varieties of Liver Suppurations 15

Intemperance in Eating and Drinking Potent Predisposing Causes to Liver Diseases in all Countries and all Climates 18

Embolisms a Cause of Hepatic Abscesses 26

Chills 27

The Cause of Inflammation and Suppuration of the Liver as Deduced from Indian Hospital Statistics ... 30

Effect of Climate on Liver Affections 33

„ Sea Breezes 38

„ Inland Situations... 40

„ River-side... 41

„ Hill Stations 42

„ Latitude and Longitude... 43

„ Rainy and Dry Situations 46

„ Atmospheric Humidity 49

PAGE

Theory of the Exciting and Predisposing Causes of Hepatitis and Hepatic Abscesses 51
Effects of Locality due to Germs 56
Causes of Liver Suppurations 62
Relative Frequency of Hepatic Abscesses in England and India 63
Diagnosis of Inflammation of the Liver 64
Signs and Symptoms of Peri-hepatitis 66
„ „ Sympathetic Hepatitis... ... 66
„ „ Malarial „ 67
„ „ Syphilitic „ 68
Treatment of Inflammation of the Liver... 69
Puncturing the Capsule of Glisson 70
Hepatic Phlebotomy 72
Atrophy of the Liver—Cirrhosis 80
„ „ Causes 80
„ „ Varieties 82
„ „ Diagnosis 83
„ „ Difficulties in the way of ... 84
„ „ Symptoms 85
„ „ Tyrosin and Leucin in Urine ... 87
„ „ Treatment 88
Ascites and Dropsy 90
„ „ Their Pathology 90
Quantities of Serum Secreted 91
Differential Diagnosis of Renal and Cardiac Dropsies ... 93
Treatment of Ascites 94
Operative Procedures 97
Embarrassments from Tumours, &c. 100
Mistakes to be Avoided 103
Hæmorrhages in connection with Liver Diseases ... 105
„ from Biliary Concretions 105
„ „ Hydatids 106
„ associated with Ascites 107, 111
, from Anus, Uterus, and Bladder 107

PAGE

Hæmorrhages from Cerebral Apoplexy 108
„ „ Apoplexy of Liver 109
„ Invisible, yet Fatal... 110
„ Epistaxis 111
„ Hæmatemesis 112
„ Jaundice the result of 112
„ Cause of Death, may be indirect... ... 113
„ Nose-bleedings in Early Life 116
„ Infantile Umbilical 117
„ Treatment of 118
Hepatic Abscesses 125
„ „ Pathology 125
„ „ Symptoms 128
„ „ Treatment 130
„ „ Illustrative Cases 131
Index 141

HEPATIC ABSCESS.

———o———

If it be true, as is frequently asserted, that from the majority of men being as mediocral in learning as in inherited intelligence, they speak of those of their contemporaries whose ideas are in advance of their time as lunatics with as much complacency as they brand as fools the unfortunates who lag behind them in thought. It might not be altogether uninteresting to know in what light the still-stands in our own profession speak of those of their pioneer contemporaries who have within the last few years not alone remodelled, but actually completely revolutionised the whole fabric of Hepatic Pathology, from its apex to its base. All familiar with recent liver-literature are aware that within the last year or two a far greater change has taken place in our knowledge of the diseases of that organ than of those of any other individual organ of the body. No practitioner, indeed, who has reached the middle period of life can fail to appreciate this fact when he casts a retrospective glance on the defective teachings he received in his student days regarding both the diagnosis and treatment

of even the commonest of liver diseases. He must well remember how he was taught to regard jaundice as a disease, whereas he now knows it is like pain, a mere symptom of many different and totally independent morbid states.

The subject of gall-stone, again, up to within a very few years ago, he must know was the bugbear of the practitioner. It was a veritable *pons asinorum* of diagnosis, from the signs and symptoms of them being so imperfectly understood that not one in a hundred of even our cleverest diagnosticians could ever feel perfectly certain whether the case he had in hand was actually one of biliary concretion or not. Moreover, at college he was told that gall-stones were in this country rare, as well as harmless, whereas exactly the contrary has been ascertained to be the case ; for since their true signs and symptoms have been recognised it is found that among the well-to-do portion of our population they are not only exceedingly common, but often hazardous to life. And there is but little wonder that in his own practice he did not make this discovery himself, seeing he had been erroneously taught that all cases of gall-stones were associated with a yellow skin and paroxysmal pain, as well as with bilious urine and pipeclay-coloured stools. While so far from this being the truth, it actually happens that in some of the most fatal forms of gall-stone affections—and they are by no means few—at no period of their career is a single one of those formerly so called pathognomonic signs ever present. Indeed, it is now well known that by far the most deadly set of gall-stone cases are those in which the concretion ulcerates its way directly out of the gall-bladder, without having entered a bile-duct at all, and never in an uncomplicated case of this kind by any chance whatever is either paroxysmal

pain, saffron-coloured urine, pipeclay stools, or jaundiced skin to be met with. (*a*)

Another inaccuracy we were all taught was that jaundice was one of the signs of hepatic cancer. Yet, strange to say, in 94 per cent. of the cases of hepatic cancer jaundice is only conspicuous by its absence.

Probably, too, some of my readers may be aware how much less common it is now-a-days than it was formerly to hear clinical teachers in the hospital wards euphemistically speaking of "obscure diseases of the liver." Just as a superfluity of prescriptions is certain proof of unsuccessful treatment, the term "obscure disease" is an equally infallible sign of non-successful diagnosis.

Further, it has only been recently ascertained that biliary concretions are not alone a frequent exciting cause of abscess, but likewise of cancer of the liver, and organic disease of the pancreas. Also, that not only biliary sand, but actual gall-stones, are no uncommon causes of both the fretfulness and convulsions of infants at the breast. And that, too, when there is no jaundice, from the concretion being impacted in the cystic duct, in which position it never causes a yellow discolouration of the skin. Added to which it may possibly surprise some of my readers to be told the unwelcome truth that it is not at all improbable that they have unconsciously done nothing to retard the progress of some of their patients to the tomb, from their having failed to recognise that a biliary concretion was the immediate cause of a hæmatemesis, a melæna, or a hæmaturia, an enteritis or a peritonitis. From the stone having ulcerated its way into the stomach duodenum, transverse colon, pelvis of the kidney, or peri-

(*a*) A full explanation of this is given in the author's pamphlet on Sounding for Gall-stones.

toneum. Or they may have allowed their patient to succumb to a curable collapse from gall-stone, from having mistaken the case for one of apoplexy, which it closely simulates, except in history and in the absence of stertorous breathing.

And what will no doubt astonish some of my readers still more will be to be told that, although their patients may have died, and post-mortems been made, not a vestige of the lethal gall-stone may have been detected by them after death. From the simple fact that nothing remained behind of the gall-stone except its mere track, sufficient to indicate to the eye of the experienced alone the course it took during the brief duration of its life-destroying career. In many cases the visible track left by the gall-stone has been attributed to something else —a perforating ulcer, for example, and wrongly recorded in the post-mortem book, from its true cause having been misunderstood, as well as its effects misinterpreted.

The cause of the error is very easily accounted for, and readily appreciated when it is made known that many a death-dealing gall-stone resembles in its course of procedure many a death-dealing rifle-bullet, which, after having effectively performed its fatal office by piercing its victim's brain, heart, or abdomen, quits the body and goeth whither no man knoweth, leaving no trace behind except its track.

Another important step in advance is the recognition that not only contagious jaundice, that is to say, yellow fever, is like some forms of hepatitis, the direct result of disease germs, but that it has its prototype in the so-called acute atrophy of the liver of this country.

These are not the only novelties the pioneers in hepatic

pathology have taught us. Some of their discoveries in the structural formation of biliary concretions are equally important as well as interesting in the extreme. For example, gall-stones have been found in oxen so closely resembling Oriental pearls in appearance as to be utterly indistinguishable from them by the naked eye. And last year at the meeting in Canada of the British Association for the Advancement of Science I demonstrated the curious fact that just as there exists a continuity in development of organised beings, so there exists a uniformity in the construction of certain non-organic concretions, irrespective alike of their chemica composition, mode of formation, or locality of deposition. This was successfully done by exhibiting a beautiful pure cholestearin radiating crystalline gall-stone (from which a lady died) of identical structure to a mass of radiating crystalline kidney-iron-stone, to a portion of Wavellite rock (phosphate of alumina), to a Derbyshire stalactite (sulphate of baryta), to a Siberian nodule (phosphorite of lime), to a marcasite-ball (sulphuret of iron), to an oyster pearl (carbonate of lime), and even to a so-called thunderbolt (iron pyrites) picked up on the chalk cliffs of Dover.

Even here our advance in liver knowledge has not stopped; for if we turn to the department of treatment, we find marvellous strides have been recently made. We now relieve our patients by piercing their liver's capsules in congestive hypertrophy. We kill their hepatic hydatids by a single puncture with an instrument not thicker than a knitting-needle. We cure acute inflammation of the liver by direct hepatic phlebotomy; instead of by the old-fashioned and but little satisfactory and roundabout modes of cupping and leeching the abdominal parietes. We empty their gall-bladders of biliary sand, gravel, and

small calculi by kneading. (*a*) We wash out their hepatic abscesses. We sound their ducts for impacted gall-stones, and when the stones are too big to pass along them into the intestine, remove them by the knife.

Having said enough, I think, to show that our knowledge of liver diseases is an entirely different thing now-a-days from what it was twenty, ten, nay, even five years ago, I now turn to the subject of these papers; and although I do not expect to be as successful here as I was in the case of either jaundice or gall-stones, in as far at least as the question of causation is concerned, from the data at my disposal being less conclusive. Yet I think I may venture to assure the reflecting portion of my readers that they will have no cause to regret honouring me with their attention. For I can at least promise them that the hospital statistics which I am about to cite will not alone reveal some startling clinical facts, but at the same time give some etiological surprises, even to those most familiar with the pathology of hepatitis and abscess of the liver. Not the least startling of which will be that, contrary to the long believed and every day taught opinion, abscess of the liver, taking all its forms together, is not only by no means a rare disease in England, but is actually more common among the poor of our metropolitan population than among either our native or our European troops stationed in India.

(*a*) As probably some readers may never have heard of my process of kneading, which I have successfully practised during the last few years, not only in the case of biliary sand and small gall-stones, but likewise in renal calculi impacted in the ureters, I may as well mention that it simply consists in forcing the concretions in the one set of cases along the bile-ducts into the intestines, in the other along the ureters into the bladder, by judiciously regulated digital pressure. The only cases in which kneading is counter-indicated being those in which there is a tendency to ulceration of the ducts.

That abscesses of the liver among the well-to-do are, for special reasons, much more frequent among Europeans living in the tropics than in England, I see no reason to doubt. But that suppuration of the liver is as rare in England as is generally believed I have good grounds to question; and I think the following are excellent reasons for the origin and prevalence of this most erroneous belief:—

1. From our not having been clearly taught the signs and symptoms of hepatic suppuration, we fail in many instances to recognise them when they are actually before our very eyes.

2. From the majority of the signs and symptoms of liver abscess not being limited to that organ, and those that are limited to the liver itself being indefinite, the hepatic suppuration is frequently mistaken for something else, more especially for the complication with which it chances to be associated. Thus, pneumonia of the right lung-base is a frequent concomitant of inflammation of the liver, whether running on to suppuration or not. And from every practitioner being familiar with the definite and unmistakable signs of pneumonia they are at once recognised. The case is diagnosed as one of uncomplicated lung disease, and the liver mischief (which may be the exciting cause of the pneumonia) escapes notice. Only those who have given special attention to this matter can have the faintest idea of how frequently such cases occur. I could mention many instances. Even this very afternoon (Dec. 19, 1885) I saw an example of the kind at Stoke Newington, along with Dr. Bartley, which is well worth alluding to. The patient, a gentleman, æt. 41, a free liver, immediately after the General Election for Parliament that has just taken place, and in which he had taken a very active part, consulted Dr. Bartley for a

pain in his right side, over the liver and lower part of right lung. Whether the liver or the lung was the first, or the most affected, it was impossible to say. From the fact that the patient, during nearly a month before the election, had not only been living very freely, but exposing himself in all weathers, and was in such a state of mental excitement that he paid no heed whatever to the state of his health, consequently he at first gave a most imperfect and even contradictory account of the commencement of his illness. When I saw him to-day, however, there was no longer room for mistaking the nature of the case. The lower half of the right lung was solid, and the liver enlarged and tender on percussion, with a distinct hepatic friction sound audible in the region of the diaphragm. Dr. Bartley's Indian experience having led him to suspect incipient suppuration, I was called in with the view of deciding the question of the propriety of immediately puncturing the liver; and it was only in consequence of the symptoms having ameliorated within a few hours of my arrival that the operation was postponed, and blistering had recourse to in its stead. The liver complication in this case, from being masked by the pneumonia, would, I have no doubt, have been entirely overlooked had the patient not had the advantage of being attended by a gentleman familiar with liver disease, and I should not have had the chance of here referring to it as a good example of the truth of the above statements.

Again, when actual suppuration of the liver has occurred, if it point, as it often does, upwards, and bursts through the diaphragm, the pus is poured into the pleura, and from its presence there being readily detected, should the history of the case be imperfectly known, it is at once diagnosed and treated as an ordinary empyema.

And this all the more readily, too, should the preceding hepatic friction sound have been mistaken (as it not unfrequently is, from being consonant with respiration) for one of pleurisy.

If, on the other hand, as often occurs in the case of small abscesses of the convex surface of the liver, the lung has become adherent to the diaphragmatic pleura and the abscess discharges itself by a small opening into the lung, from cough and expectoration being present, a purulent bronchitis or pneumonia is, instead of the hepatic abscess, not unlikely to be diagnosed. While writing this paper I received a note from Dr. Gandy, of Norwood, telling me of the death from suffocation of a gentleman, æt. 43 (from U.S.A.), who refused to allow us to puncture his liver a few days ago. The result has been that the liver abscess has burst into the lung—a by no means uncommon accident (*a*)—and thus without a post-mortem revealed the existence of the hepatic suppuration.

3. From the general being still less definite than the local signs and symptoms of liver abscess, they of themselves, in like manner, oftentimes lead to a mistaken diagnosis. Not unfrequently the case is thought to be either one of typhoid, blood poisoning, or hectic fever, in consequence of there being rapid pulse, high temperature, diarrhœa, abdominal tenderness—without any distinctly local hepatic pain—rigors, sweatings, and nocturnal exacerbations. From each of these symptoms being more or less marked in different cases they lead, according to whichever chances to be the most prominent, to one or other of the above-mentioned erroneous diagnoses. Cases of abscess of the liver have even been admitted into, and

(*a*) See p. 826, author's work on Liver Diseases.

treated in hospitals as cases of typhus fever, and the mistake only been discovered at the autopsy.

4. Another reason why we have remained so long ignorant of the fact that abscess of the liver is by no means unusual amongst us has arisen from the circumstance of its being most commonly met with among the rich—the class in which autopsies are the least frequent—and as the case, either on account, or in spite of its inappropriate treatment, often ends fatally, and without a post mortem, the cause of death is recorded under a wrong name, and our mortality tables fail to tell the tale of the frequency of hepatic suppurations amongst us.

With these not unnecessary preliminary remarks, I now enter upon the subject of abscess of the liver and its antecedent hepatitis.

That few questions in medicine require so complete a revision as that of the cause of suppuration of the liver tissues has just now received ample confirmation by the appearance of an article on the subject by Surgeon-General Moore, (*a*) who, while attempting to upset some of my statements regarding the predisposing causes to liver abscess among Europeans resident in India, has clearly shown by his theories how very imperfectly understood is the question of the causation of hepatic suppurations in the tropics even by some of those who presume to write with the authority of an extensive personal experience of the subject.

As the prevention rather than the cure of disease is now occupying the minds of the greatest of our medical reformers, and there can be no doubt that a knowledge of the causation of a disease is by far the most direct

(*a*) "Cause of Hepatic Abscess." By Surgeon-General Moore. *Lancet*, Oct. 31, 1885, p. 798.

road leading to the discovery of a means of its prevention, I think I cannot do a greater service to the profession than by going as fully into the question of the causation of the disease now under consideration as the data at my disposal admit of.

And as Dr. Moore's mode of criticising my views of the cause of the prevalence of abscesses of the liver in India—he being himself a Bombay medical officer—conclusively shows how little is actually known regarding the intricacy of the question of the causation of liver suppuration, it is probable that my object will be best accomplished by commencing the exposition of the subject with a critical analysis of his statements as far as they go. For I will thus be enabled to place the question before the reader in a more impressive light than I should otherwise be able to do.

Before beginning, however, to criticise the criticisms of my critic I must remind the reader that all suppurations of the hepatic organ are neither identical in their origin nor in their precise nature, and that they naturally divide themselves into three distinct varieties, which for the sake of clinical convenience I have respectively named Idiopathic, Traumatic, and Metastatic. It is necessary also to bear in mind that, notwithstanding that these three names look so definite and independent on paper, there is in reality no hard and fast line of demarcation to be drawn between any of them. On the contrary, the three varieties not only run by imperceptible degrees into each other, but it is actually in many instances—logically speaking—impossible to say to which variety a particular case of hepatic abscess belongs. The cause of the difficulty is not far to seek; for although not only the exciting, but even the predisposing causes of the three varieties be not identical in as far as their morbid histo-

logy is concerned, their nature is the same; for precisely similar pathological processes take place in them all, although the *fons et origo* of the suppurations is in each different. I desire it still further to be borne in mind that all three varieties are met with in every country of the world, though their relative frequency is greater or less in certain places, according to the specific differences of the climate and the locality. The first-named variety predominating in tropical, the two latter in temperate zones. Everywhere, however, they are equally difficult in diagnosis, uncertain in treatment, and fatal in results.

As I am not at present writing with the view of teaching medical students the rudiments of hepatic suppurative inflammation, but with the object of furnishing my medical brethren, who are already acquainted with the subject, with some new data to act as fresh food for mental digestion, there exists no necessity for taking up time by going into the causation of either the traumatic or metastatic varieties of liver abscesses, as their titles sufficiently denote their origin. I shall therefore at once endeavour to throw a new light on the probable predisposing and exciting causes of the idiopathic variety of liver suppuration, and its antecedent hepatitis. I do this all the less reluctantly from the fact that, as will be seen in the sequel, the very mystery hanging around its etiology has to the thinking mind a special charm about it.

Fault-finding never being an agreeable occupation, and the biter being occasionally the worst bitten, it may be judicious for me to preface my strictures on Dr. Moore's observations with an acknowledgment that I am not unconscious of the fact that from its being invariably much easier to detect the weakness than to determine the strength of an opponent, it is wise, ere one dips his

pen into the unsavoury ink of controversy, either with the view of attacking or of defending a debatable proposition, to weigh his own weakness, as well as his strength, in the same scales as he does that of his opponents. Otherwise it may happen that he unwittingly allows to escape unperceived some vulnerable point in his mental armour through which he may be dealt an unexpected blow, the very unexpectedness of which rendering it all the more mortifying. This remark emanates from my pen in consequence of its being apparent that, while Dr. Moore is undoubtedly justified in considering that his long service in India and wide experience of Anglo-Indian diseases admits of his speaking with authority on the question of the causation of abscess of the liver, he appears to have altogether lost sight of the fact that a physician who has never put foot on Indian soil, but who has for years made liver diseases a special study, and been in the habit of seeing and treating cases of them from every quarter of the globe has an almost equal right to have his views listened to with respect, and as fairly considered, as one whose plea to be heard consists only in his having had the advantage of a personal experience in a circumscribed area. Even though the sphere of his labours may have been the very one embraced in the controversy.

Another cause for complaint exists in the fact that, as most of the statements set forth by Dr. Moore as condemnatory of my views are not alone nothing more than reiterations, though in different language, of my published opinions, but even the statistics he adduces to condemn them in reality confirm, as I shall presently show, the exactitude of my inferences, I consider he was scarcely justified in so strongly objecting to my opinion.

Gluttony and Intemperance as Predisposing Causes of Liver Suppurations.

After kindly designating my book, in which the views he objects to occur, as an admirable one, Dr. Moore asserts that I have most erroneously said that gluttony and intemperance are two of the main causes of the frequency of hepatic abscesses in European residents in India. Most willingly do I admit the truthfulness of the impeachment. And not only so; but I will even be so bold as to go a step further and unhesitatingly add that these two factors are not alone the most common of the predisposing causes of idiopathic suppuration of the liver in India, but everywhere else. And this is an important statement to bear in mind, seeing, that idiopathic abscesses of the liver are limited to no country, no clime, and no people on the face of the earth. Europeans, Indians, Africans, and Americans—North, South, and Central—all rendering themselves equally liable to be attacked with suppuration of the hepatic organ when they consume more food and drink more spirituous liquors than their systems can assimilate with advantage. The mere presence of an exciting cause, of which there are many in every quarter of the globe, when the predisposition exists, being all that is requisite to call the inflammation and subsequent suppuration into existence.

After so positively denying the truth of my statements regarding the much greater frequence of liver abscesses among the well-to-do Europeans in India (my remarks had special reference to them) being due to the habitual over-indulgence in rich food and strong drink, coupled with an inactive mode of life, strange to say, Dr. Moore

remarks that no one seeks to deny the observations of Macnamara, who "long since demonstrated that the mode of life of soldiers, especially in former days, was conducive to fatty liver and hepatic abscess." Such being the case, why, then, I ask, does he complain of my having given publicity to the same ideas? Here is his answer:—

"I entirely deny that the habits of Europeans in India are generally such as portrayed by Dr. (George) Harley. Gluttony and intemperance are no more the characteristics of Anglo-Indians than of similar classes at home." This, to my mind, is scarcely a fair way of presenting my statements to the reader. Firstly, because it incidentally implies that I accuse all Anglo-Indians of gluttony and intemperance, which I decidedly do not. All I say being that gluttony and intemperance are fruitful sources of liver abscess among the well-to-do Anglo-Indians resident in India, be they military or be they civilians. And even that statement I most guardedly qualified by an explanation of what I meant by the words gluttony and intemperance, which qualifying part Dr. Moore unfortunately seemingly accidentally omitted, and consequently gave an entirely exaggerated philological signification to these words as used in my book. What I really say is (page 809): The chief exciting causes of hepatic abscesses are gluttony and intemperance, which, in proportion to the habits of life, are far more common in Europeans living in the tropics than in the same class of individuals resident in temperate zones. It is to be remembered that the words intemperance and gluttony are here employed as relative terms, ruled and modified by collateral circumstances, as will presently be explained." Now for the explanation.

As circumstances alter cases, so do the influences of temperature, locality, and occupation change the intrinsic

value of words when applied to the quantity as well as to the quality of the food and drink consumed by an individual in a given time. For an amount of either one or the other, or of both, which might be justly regarded as moderation under certain conditions, might with equal propriety merit the title of gluttony and intemperance when indulged in by the same person placed under different circumstances and pursuing a different mode of life in another locality. A self-evident proposition, I should imagine. But in order to prevent all possibility of a repetition of a mistake being made in my meaning, I will digress for a moment and remind the reader of the well-known fact that a man who, while living in London, may be both a moderate eater and drinker, not perhaps from choice, but from constitutional necessity, may no sooner find himself on the bracing moors of the Highlands of Scotland, than he eats and drinks, not alone with impunity, but with actual advantage, a quantity of food and an amount of whisky which would, if taken by him in London, make him actually ill. And justify the terms gluttony and intemperance being applied to him. Hence we see that what would be justly regarded as moderation in eating and drinking under one set of circumstances might with equal philological and philosophical justice be stigmatised as gluttony and intemperance in others.

A precisely similar rule of comparison holding good in the case of Anglo-Indians, I say that those of them who indulge their appetites with the same daily quantities of food and spirituous liquors in India as they with perfect impunity do while in England, may with propriety be said to be guilty of gluttony and intemperance. From the wants of the system, as well as its capabilities of assimilation being quite different in a hot and in a cold country.

Seeing that it requires but a very elementary physiological knowledge to be aware of this, and I cannot possibly attribute any deficiency on this score to Surgeon-General Moore, the only way I can at all account for his objecting to my statement is by supposing that he attaches a far more vivid signification to the words gluttony and intemperance than I do. In case this supposition be correct, I will here subjoin for his consideration the dictionary meanings of these two English words. In Webster's large and unabridged edition, which I think will be accepted as an excellent authority on the point, gluttony is defined as an "excess in eating; extravagant indulgence of the appetite for food." Now, what does an excess in eating mean? Nothing beyond that one eats more than the wants of the system demand. And if Anglo-Indians while residing in India eat anything approaching the quantity of rich hydro-carbon foods as they do whilst living in England, will anyone, I ask, have the foolhardiness to deny that they eat in excess? Unless he be prepared at the same time to assert that they take more muscular exercise in India than in England. In order that all the hydro-carbons (which the system has but little use for in hot India) may be burned off. I put this question emphatically. For we not only know that if all the hydro-carbons taken into the stomach be not daily consumed, the residuum by accumulating in and clogging the *prima via*, deteriorates the constitution. But we also know that it is particularly that organ whose especial duty is their dispersion—namely, the Liver, which suffers.

As regards Webster's definition of the word Intemperance, again. It is: "The habitual indulgence in drinking spirituous liquors, with or without intoxication." Here, be it noted, it is not put down as synonymous with

drunkenness. On the contrary, a person may be decidedly intemperate, and yet no drunkard. I have known, not one, but several individuals who are very intemperate, in the dictionary sense of the word; for they admittedly take what they euphemistically call "nips" at all hours of the day. Yet they assert—and probably truthfully—that they have never in their lives been actually intoxicated. Seeing that to be intemperate, then, neither necessitates nor implies actual drunkenness, I cannot see why Dr. Moore has been so irate against me for employing the word. Especially, too, as he must be fully aware, that the indulging in "nips" between meal-times by persons with a predisposition to liver disease, either hereditary or acquired, through the enervating influences of climate, is universally admitted to be a most prejudicial one. Indeed, I fear that, no matter what he may say to the contrary, unless he can give me proof that I err, I shall still consider I am justified in stigmatising this habit of taking nips between meal-times as intemperance when indulged in, as it often is, in various parts of British India. Moreover, my assertion that an over-indulgence in eating and drinking is a source of Indian liver abscess by no means implies that I accuse all Anglo-Indians of gluttony and intemperance any more than I accuse our own inhabitants of the same propensity. To so interpret my words would not be one whit less illogical than to assert that because steel grinding is a fruitful exciting cause of phthisis in some of our manufacturing towns all the inhabitants of these towns are steel grinders. A palpable absurdity, of course. But not more of an absurdity, to my way of thinking than the rendering the Surgeon-General has thought fit to give to my statement that gluttony and intemperance are among the chief predisposing causes of abscess of the

liver in Anglo-Indians. I even doubt not that I am quite as cognisant of the fact as he is that there are not only many total abstainers among Anglo-Indians, and even some rigid vegetarians, who rather under than over feed themselves, but likewise, as he states (as an apparently little known fact), that "during the last quarter of a century a greater change has taken place in Anglo-Indian habits of life than even in home habits of life, and Anglo-Indians as a general rule neither eat, drink, nor sleep to excess." The latter assertion is undoubtedly true, for there exist in India two most potent enemies to sleep—heat and mosquitoes. But why he should have taken the trouble to prominently call my attention to the improvement that has taken place in Anglo-Indian habits I am quite at a loss to understand, seeing that what he says is merely a reiteration of what I wrote at p. 242 of my book. Aye, and that, too, I think, in quite as forcible language as he employs. Here are my words:

"At one time nearly one-half of the liver cases coming home to this country from India were directly traceable to an habitual over-indulgence in rich foods and strong drinks coupled with an indolent mode of existence. Fortunately for the livers of Europeans inhabiting the tropics the fashion of drinking strong bitter 'Indian ale,' at all hours of the day, has given place to the less dangerous habit of consuming light French clarets. While at the same time luxurious sofas and wicker lounges have been in a measure abandoned for lawn tennis and football. And as a natural result not only are much fewer 'Indian liver' cases now met with in London, but the cases that are met with are, in general, of a much milder type than they were between twenty and thirty years ago. Another reason for this change, however, may be that since the communication with India has become so

much easier men run home oftener, and when they get ill, as a rule, they return home earlier, and thus greatly diminish the dangers of Indian service. I am sorry, however, to be forced to add—from my own personal observation—that Indian liver cases are still much more common than they ought to be, or would be, I imagine, if more attention were paid to food, drink, and exercise, For careful inquiries among the Indian liver patients who come to me have led me to the conclusion that, notwithstanding the improvement that has taken place in the habits of Europeans resident in the tropics, there is still room for more. As one and all confess that there is still prevalent an habitual over-indulgence in rich, highly seasoned, stimulating food, both by men and women, while resident in climates so hot as to render it impossible for them to take sufficient bodily exercise to use up all the hydro-carbons admitted into the circulation."

This language appears to me to be explicit enough to have prevented any ordinary reader misinterpreting it.

It may probably not have escaped the reader's attention that while endorsing everything that even the most favourably disposed can say regarding the vast improvement that has in recent years taken place in the habits of Anglo-Indians, I nevertheless insinuate that there still exists room for improvement. As it is possible some one may feel dissatisfied with this suggestion, I venture to inquire of him if, throughout India as a whole (for customs are different in different parts) even yet between meal times there has entirely ceased to be heard within the precincts of Anglo-Indians' (both military and civilian) bungalows calls for delectable "pawnies," "pegs," and "pick-me-ups?" And further, if the mixtures known by these names have become disassociated

from stimulating beverages. Such as brandy and soda, sherry and bitters, and, what is worse still, that liver-bane, sour champagne, presented to the unwary under the disguised titles of "très-sec" and "brut"? If he can assure me that they are, I shall be delighted to allude to such an important fact in my next editions. Another of Surgeon-General Moore's statements which appears to me not only to be a totally uncalled-for one, but a most unaccountable bill of indictment, when its preamble is considered, is that he, after citing the important statement of Macnamara, that spirit-drinking is conducive to fatty liver and hepatic abscess (which, instead of being in opposition to, is quite in confirmation of my views), assigns as his reason for objecting to what I say regarding the causation of liver abscess that "hepatic abscess cannot, however, be ascribed altogether, or even in the greatest degree, to such causes." Thereby implying that I had done so. While, so far from such being the case, I not only begin the chapter on Abscess of the Liver by saying—"Suppuration of the liver may accrue from a great variety of exciting causes, in any country or climate, as well as at any period of life between early infancy and advanced age." But further on, in the very same chapter, make the following statements (p. 807): "That of the multitudinous and varied exciting causes hepatitis is the most common producer of hepatic abscess." While among the causes of hepatitis I mention (p. 806) gall-stones, inspissated bile, dysenteric and other forms of intestinal suppuration, as well as embolism and climatic malarial influences. Consequently, as hepatitis, in one form or another, invariably precedes hepatic suppuration, it naturally follows that abscesses in the liver have the same exciting as well as predisposng causes as hepatitis.

Passing from this, to me necessitated though distasteful, criticism of a critic, I joyfully avail myself of the opportunity of calling attention to what the Indian Government's Reports tell us regarding the enormous decrease of mortality which has taken place among our European troops during the last twenty years, as it has a most important indirect bearing on the question under consideration. For the decrease in the death-rate, if not greatly dependent upon, is at least coincident with, the improvement that has taken place in the Indian habits of the European troops.

Professor Maclean says that the mortality in the European part of our Indian army has actually diminished from 79 per 1,000 in 1860 to 13·07 per 1,000 in 1882! And the reports show that the deaths from hepatic abscesses have diminished in a like ratio.

I do not wish it to be imagined, however, that I attribute all this sanitary improvement to mere changes in the dietetics and the modes of living of our troops. For I shall presently show the vast importance of locality upon death-rate from the liver diseases affecting Europeans in India. By which it will be apparent that the changes that have been made in the situations of some of the cantonments must have had a material influence in bringing about the greater healthiness of the troops. I now come to the consideration of Surgeon-General Moore's suggestion that

Embolism is a Direct Cause of Liver Abscess.

Dr. Moore apparently propounds the idea that embolism is a cause of hepatic abscess as a novelty. I fear, however, I must call his attention to the fact that, like

many of his preceding considered statements, it, too, is but another reiteration of what I have myself said at page 838 of my book, and which I take the liberty of asking him to reperuse. The point on which we differ consists in his thinking that embolisms are a common cause of liver abscess, and due to chills in anæmic persons.

To this view I object—firstly, because of all the organs of the body, with the single exception of the spleen, the anastomosis of the blood-vessels in the liver are about the freest. And secondly, I have yet to learn why a chill would be more likely to cause an embolism in a blood-vessel of the liver than anywhere else in an anæmic subject. On this point, however, I shall be glad to have further information.

Chills as Exciting Causes of Liver Inflammation.

Chills being one of the most fruitful causes of all kinds of inflammatory affections, in various organs of the body, one cannot feel surprised that the liver is not exempted from their attacks. Chills, indeed, in the predisposed, are an exceedingly common exciting cause of hepatitis, and were it not, as I before said, that a thorough understanding of the etiology of inflammatory liver affections may be the surest as well as the most rapid means of our arriving at their prevention, I might at once pass on to the consideration of the next exciting cause. From Dr. Moore's having, however, unfortunately made some statements regarding hepatic-chill action in India, which are unsupported by clinical experience, it is necessary for me, in order to prevent the propagation of what I consider to be an error, to comment upon them. To avoid mistakes I shall cite his words. They are: "I believe liver inflam-

mation and abscesses are usually simply the immediate result of atmospheric vicissitudes, nowhere so powerfully felt as in India, where on the coast the diurnal sea-breezes succeed to a hot, moist, stagnant atmosphere, causing a sudden and considerable fall of temperature daily, where throughout the country during the whole year the night temperature . . . is so much less than that of the day, . . . where the cutaneous surface of Europeans is rendered extraordinarily susceptible to lowering of temperature by the over-excitation and consequent cutaneous debility produced by heat, and where Europeans, and natives who neither eat nor drink too much expose themselves habitually in the most utterly careless manner, after spasmodic exertion, to that fertile source of most diseases, and especially of liver disease—chill . . . chill induces in the robust, as a first step, a congestion or inflammatory condition, and in the anæmic probably hepatic embolism, the result being abscess." In this it appears to me that he fails to perceive that in presenting this theory he is really giving us a nut without a kernel. The why and the wherefore of the *liver* being the special point of attack of the chill in India not being even so much as touched upon.

Every one is aware that vicissitudes of temperature occur, and that chills are a frequent cause of hepatitis, the precursor of liver abscess, in the predisposed in all countries—temperate, as well as tropical—consequently, I am at a loss to understand why he has thought fit to put down a chill thus prominently, as if it were a specific cause of Indian liver abscess—without, at the same time informing us why an Indian chill specially attacks the liver. I should imagine that, as the effects of chills on the human body are nearly the same all over the world, if,

as he asserts, they are a special cause of suppuration of the liver in India, there must exist some equally special predisposing cause in that organ in India, to make the chill from an Indian vicissitude of temperature select it for its attack in preference to any other of the internal organs, such as the lungs, the kidneys, or the spleen. Without some explanation of why the liver is specially selected by the chill in India, Dr. Moore has, I opine, presented us with a pathological nut, minus the kernel. And, consequently, his theory is of no avail in deciding the question of the special cause of liver abscess in India. I would further remark "en parenthese" that while he entirely omits to recognise the fact that there are quite as great diurnal vicissitudes of temperature in other parts of the world as in Hindustan, without their being accompanied with the same prevalence ot suppurative inflammation of the liver among the Europeans inhabiting them, he has equally ignored the fact that there are many places in more northern zones where the seasonal, as well as diurnal variations in temperature are quite as great, if not even greater, than any met with in India. Even in Europe we find places such as Moscow, where between the summer's heat, and the winter's cold there is no less than 82° of difference. While in London it is only 57°. Facts not to be disregarded when considering a temperature theory of the causation of Indian liver abscess. As the fact of such significance being given to vicissitudes of temperature by an Indian medical officer of Dr. Moore's professional standing, and no notice whatever being taking of the "predisposing" causes to abscess of the liver among the European residents in India; or even so much as a distinction being drawn between special Indian predisposing and general (in all countries) exiting causes appears to indicate that little is known of the valu-

able information furnished by the Indian Government Reports. I shall now adduce some data which were kindly extracted for me from them a couple of years ago by Surgeon-General Edward Balfour, well known as the author of the Encyclopædia of India, as well as the compiler of some of the reports on the Barometrical Sections of India. Although Dr. Balfour, is of course, accountable for the exactitude of the extracts, I hold myself responsible for the truthfulness of the deductions drawn from them and the calculations grounded upon their figures, as well as for the changes made in their arrangement. The facts and statistical figures themselves are, however, nothing more than impartial extracts from the "Report on the Vital Statistics of the European army in India (*a*) during the ten years, 1870 to 1879 inclusive.

Indian Hospital Statistics, bearing on the Causation of Suppurative Inflammatory Affections of the Liver.

As the statistics about to be put before the reader furnish data directly opposed to any mere temperature theory, as a direct exciting cause of hepatic abscess, as well as reveal the altogether unexpected and important fact, that "circumscribed localities" have a far greater influence on the deaths among Anglo-Indian soldiers from inflammation of the liver than either temperature, latitude, longitude, altitude, rainfall, or any other of the ordinary so-called climatic influences, either separately or

(*a*) The report was drawn up by Surgeons-Major Bryden and Stephen. I should have been much better pleased could I have given statistics of the rates of mortality of all Anglo-Indians, civilian, as well as military, but as there exist none of civilians one must content himself in this, as in all other instances, with what he can get.

combined. It may be well for me to remind the reader of the extent of country included under the name of British India. It actually covers a surface of no less than 1,486,319 square miles, and extends in a direct line from Cape Comorin close to the equator, 1,900 miles north to Peshawar. So that there is contained in this vast area an infinite variety of climates. Indeed, India has within its wide borders chains of mountains of such varying degrees of altitude as suffice to furnish almost every variety of nocturnal and diurnal temperature on the face of the globe. So it is absolutely necessary for the reader to bear these things steadily in view when considering the statistics of Indian climate, in connection with the causation of hepatic abscess, and its antecedent inflammation of the liver.

Besides which I must call attention to the fact that if we are content (as the sequel will show) to draw our conclusions as to the relative frequency of liver abscesses in different Indian localities, from the hospital statistics alone, we shall be led into grave error. I have therefore adopted another method of arriving at the truth, and one too, which has the double advantage of killing two birds with one stone. Before mentioning it, however, it will be advisable for me to state my reasons for declining to rely on the data of liver abscesses furnished by mere hospital statistics. They are: Firstly, certain forms of hepatic disease, such as biliary concretions, fatty liver, hepatitis, and abscess, are far more common among well-to-do people, than among the class of persons who go into hospitals. Consequently, hospital statistics of the relative frequency of such forms of liver disease yield most imperfect data to draw conclusions from, as to their absolute frequency in any given locality. Secondly, it being an undoubted fact that, from its not always being an easy

matter to diagnose the existence of pus in the liver, many cases of death from liver abscess are attributed to other causes, it would be hazardous policy to rely implicilty on abscess statistics. This is proved by the fact that not alone in India, but in every other country hepatic abscesses have again and again been discovered at the autopsies of persons in whom during life the existence of purulent matter in the liver was never even so much as suspected. These reasons, I think, not only justify me in objecting to place reliance on hospital statistics, where the existence in, or the absence of pus from the liver has not been physically demonstrated, in attempting the elucidation of this question. But, likewise, in employing another method, which, though more indirect, has the advantage of being much less likely to mislead. This other method is based on the fact that no pus can form without inflammation. That every liver abscess is preceded by hepatitis in some form or another, and that the signs and symptoms of any inflammation of the liver sufficiently severe to have ended in suppuration of the hepatic tissue is unlikely to have escaped being diagnosed by any well-educated practitioner.

I, therefore, propose to rely on the evidence furnished by the hospital statistics of hepatitis. From believing that they are more likely to enable us to arrive at the truth of the relative frequency of liver abscess, in considering the question of its causation than anything else. Besides which, by employing the statistics of hepatitis we have the advantage of, at one and the same time, obtaining perhaps a clue to the causation of mild inflammation, as well as of severe suppuration of the hepatic tissue.

The first point to be considered in this inquiry is the influence of

A Tropical Climate as a Predisposing Cause of Hepatitis, and Hepatic Abscess among Europeans.

Having gone so fully into the subject of injudicious dietetics, in connection with climate, as a predisposing cause of liver derangements, I must now show what the Government statistics tell us regarding the specific influence of a hot climate, *per se*, as an inducer of inflammatory affections of the liver.

The general idea at present is, I believe, that a European constitution, under favourable circumstances, gradually gets more and more emancipated from the injurious influences of tropical heat, the longer it is exposed to them. In a word, becomes acclimatised. This no doubt, as a rule, is perfectly true. But, as every rule is said to have its exceptions, I have now to call attention to an unanticipated exception in this one too. The exception being in the European's *liver!* The consideration of the reason for the liver being an exception to the general law of adaptation, I shall, for a moment, put on one side, and deal merely with the fact, and the data at my disposal in its confirmation.

In the first place, the fact must, I think, be regarded as undeniable, when the following Indian Government statistical data are reflected upon. For while the hospital statistics of the European regiments serving in India show:—That the amount of general sickness among the troops that have been stationed for some time in the country is smaller than among those who have only recently arrived in India; they also show that the highest rate of mortality from hepatitis is actually among the

troops who have been longest resident in the country, and therefore most exposed to the climate of Hindustan. This curious fact that the human liver should be a marked exception to the general law of acclimatisation the statistics show in no ambiguous language. For while the Report shows that during the ten years 1870 to 1879 inclusive, the hospital admission rate in the European-Indian army generally, from all diseases, was 1475 per 1,000 of the strength; that of regiments in their first year of Indian service amounted to 1612 per 1,000. It further states that while the daily sick-rate of the army generally was 60 per 1,000 that of the fresh arrivals was 64. (*a*) In the case of the death-rates from hepatitis, however, the position of the acclimatised and non-acclimatised troops are entirely reversed; for while in the army generally the mortality from hepatitis averaged 2·19 per 1,000—ranging during the ten years from 1·62 to 3·28 per 1,000—in the regiments serving for the first year in India, it only averaged 1·83—ranging during the same period of ten years from 0·68 to 3·91. While, again, it tells us that the soldiers of the regiments with longer residence, and consequently more aged troops succumbed in much greater proportion to prolonged or repeated attacks of hepatitis. That this must have been the case is, of course, proved by the fact that, while the average mortality of the whole of the English regiments amounted to 2·19, that of the troops recently arrived in the country was only 1·83 per 1,000. The actual numbers were:—

(*a*) As after the completion of the skeleton, mortality increases as age advances in all climates, the effect of acclimatisation ought never to be calculated on the relative mortality; but on the amount of relative sickness alone, as has been here done.

DEATHS FROM HEPATITIS FROM 1870-79.

	Total deaths.	DIED PER 1,000 OF THE STRENGTH.			
		Under 20 years of age.	20-24.	25-29.	30 and upwards
Bengal Army	611	0·08	0·86	1·90	3·00
Bombay „	165	—	0·81	1·78	3·12
Madras „	311	0·45	1·51	2·84	4·90

Unfortunately, no reference is made in the statistics to the probable influence of acclimatisation on the death-rate; but as the ages of the newly arrived troops generally vary from 18 to 24 years the death-rates at the different ages above given, may be considered as approximately indicative of the relative number of years of Indian residence. So it is thus seen that the liver, for some reason or another is a most marked exception to the general law of tropical acclimatisation. To me the reason appears to be palpable enough. And so, also, it ought, I think, to be to every one who has carefully reflected upon what I said was the effect upon the European's liver of over-eating and drinking. Should it, however, happen that the reader does not accept my view that the reason why the liver fails to become acclimatised is on account of its being not only daily, but weekly, monthly, and yearly over worked, in consequence of its possessor consuming more food or spirituous liquor than his liver can readily prepare for assimilation and dispersion. In a word, that the organ, as a result of prolonged ill usage, gradually strikes work, and finally breaks down. I re-

quest him, ere he ventures to express a contrary judgment regarding the cause of the liver failing to become, like all other organs of the body—acclimatised—to carefully peruse the other statistical data that I have in store for him regarding the apparent effects of the different elements out of which the influence of climate upon the European constitution is constituted. For I think he will then see that, be the influence of so-called climate —considered *per se*—what it may, there can be but one opinion regarding its enervating effects upon the Anglo-Indian's health being potently supplemented by the two factors which I have designated as gluttony and intemperance. (*a*) For there cannot possibly, I imagine, be a doubt that, if the liver be really the one solitary organ in the European's body which does not become acclimatised in the tropics, there must necessarily exist some special and specific reason why it is so marked an exception to the general law of climatic adaptation. And I can see none other.

Having finished with the consideration of what I look upon as the predisposing causes, I now turn to the special and local exciting causes of liver inflammation and suppuration in India. The first point demanding consideration in this part of the inquiry will be to try to discover, if possible, the influence of the climate in relationship to geographical situation. Hence I shall now adduce data bearing upon the Relative Mortality from Hepatitis in the Different Presidencies of India.

(*a*) In confirmation of my views regarding the all important influence of intemperance on liver affections, I beg to refer the reader to the "Supplement to Registrar-General's Report" of our National Mortality for 1885, where, at p. xv., he will find the following remarks:—"the mortality from liver disease among men engaged in the liquor trades is six times the average for males generally." See also further remarks at p. xxxvii., and tables of mortality at pp. xxxii. and lxi.

The Relative Mortality from Hepatitis in the Different Presidencies of India.

The Government statistics show that during the ten years reported upon, the strength of the army was 577,416 (the regiments being stationed chiefly in 81 different cantonments), and the admissions into hospital from all causes were 851,564. Of these 28,780 were cases of hepatitis. With a total of 1,267 deaths from that cause. And it will be considered that these numbers, as Dr. Balfour says in a letter to me, are probably of sufficient magnitude to admit of more correct deductions being drawn from them regarding the probable frequency of liver abscess, than from the more limited and less trustworthy statistics of those recorded as hepatic abscesses. The first noteworthy point to which attention is drawn by the statistics is that, in the Madras Presidency the mortality from hepatitis was 3·16 per 1,000 of the army strength. In the Bengal Presidency 2·04. And in that of Bombay only 1·71. Now, it will be asked are these numerical deviations in the death-rates due to differences in the conditions under which these soldiers served? The reply to this is: Their food, their clothing, and their duties were identical; while, as regards the factors of heat and housing, not only are they in all British India very much alike, but from the fact that the regiments were moved, at short intervals, from one cantonment to another, the advantages or disadvantages of the stations are almost completely equalised on these points. It may be added too, that from the coming and going of the soldiers under the short service system, it is probable that, during these ten years, there were, at least, three complete changes in each of the 81 cantonments. What then, it may be inquired, was the cause of this extraordinary difference in the mortality from hepatitis in the

three Presidencies, the relative proportion as just seen, being 1·71, 2·04, and 3·16? As some of my readers, who are unaccustomed with the handling of statistica tables, may scarcely realise the relative values of these apparently insignificant decimals, in connection with such an enormous number as 28,780 cases of hepatitis, in ten years, I herewith give the result as calculated at per cent., which will be more impressive to the statistically uneducated eye. It then reads thus: There are 119 per cent. more cases of hepatitis in the Bengal than in the Bombay Presidency, and 178 per cent. more in that of the Madras Presidency. These are, indeed, striking differences, and must, of course, be due to some specific, though as yet unknown, cause. What this cause may be it is now my endeavour to discover. The first point then will be to ascertain the rates of mortality at those stations which might, *à priori*, be regarded as being the freest from inherent tropical climatic influences. With this view the following table is given to show the:—

DEATH-RATE FROM INFLAMMATION OF THE LIVER AMONG EVERY 1,000 EUROPEAN TROOPS STATIONED AT SEA-COAST CANTONMENTS. (*a*)

Port Blair (Andaman Islands) Bay of Bengal	0·83	Average Death-Rate 2·44
Madras	0·88	
Kurrachee, on the Arabian Sea	1·17	
Malliapuram ,, ,, ...	2·17	
Aden ,, ,, ...	2·20	
Bombay ,, ,, ...	3·77	
Cannanore ,, ,, ...	4·11	
Calicut ,, ,, ...	4·40	

(*a*) As the omission from or introduction into a small statistical table like the above of one single high or low death-rate, would vitiate the result in calculating the average, all the sea-coast stations in the report are included.

It will be noticed that, notwithstanding that each of these eight seacoast stations may be said to be washed by the waves of the ocean, they present very marked differences in their death-rates. The two that have the decidedly smallest lying on the east; those manifesting the highest death-rate lying on the west side of Hindustan. The last three in the table are under the direct influence of the periodic south-west Monsoon. The action of the Monsoon winds, however, scarcely I think explain the nocturnal and diurnal vicissitudes of temperature theory so strongly insisted upon by Dr. Moore as the main cause of liver abscesses among Europeans. On this point, however, I wait enlightenment. And, meanwhile, as it appears to me there must be some other hepatitis-producing agent at work to explain the disparities in the death-rates at these seaside places I subjoin a table of mortalities at thirteen different inland stations scattered over the country. In order to give a fair average, and thus ascertain, if possible, the influence of the independent factor of inland situation over the death-rate, so as to fairly test the vicissitude of temperature theory, I include some stations near to, and some far from, each other, some at one height, some at another, some close to water, others at a distance from it.

Here as in the sea-coast table we find a considerable difference in the extreme death-rates. Although, on the whole, a degree of uniformity runs through them, and not only so, but, as is seen, the mean death-rate is considerably lower (1·81) than on the coast (2·44). This being the case the next point, is to try and see if the proximity of water *per se;* that is to say, independent of diurnal and nocturnal vicissitudes of temperature due to sea breezes has a material effect on the death-rate of Europeans from inflammation of the liver. So I now subjoin a table

MORTALITY FROM HEPATITIS AT INLAND STATIONS.

Place.	Situation.	Feet in Height above Sea-line	Death-Rate per 1,000 Stgh.
Jhansi ...	Lying low on the banks of a lake ...	765	0·97
Jullundhur	On open plain ...	937	1·27
Bellary ...	Away from water ...	1,300	1·28
Ferozepore	3½ miles from Sutlej	720	1·35
Belgaum ...	On the basin of a water course ...	2,240	1·45
Amratsar, Govinhdur	Away from a river ...	900	1·58
Sialkot ...	On the Surma river ..	900	1·67
Agra ...	„ Jumna ...	800	1·72
Dehli ...	„ „ ...	800	1·78
Kamptee ...	„ Kanhan ...	939	1·79
Neemuch...	Away from a river ...	1,476	1·88
Morar ...	On the Chumbul ...	800	2·80
Meerut ...	Away from a river ...	900	4·15

Average Death-Rate 1·81

of the mortality at 13 places on river banks. The three places marked * are situated at the junction of rivers.

Although this table shows a decided diversity in the death-rates from hepatitis at some of the stations, being as low as 0·27 at Hyderabad on the Indus, and as high as

4·94 at Barrackpore on the Hugli. From its average mortality rate being greater than that of inland stations (irrespective of situation) 0·42, and only 0·21 lower than that of the sea-side stations. The idea is naturally suggested that perhaps the proximity of water may be a specific influencing factor in the death-rate of Anglo-Indians from liver disease in India. Consequently, it is desirable to

MORTALITY FROM HEPATITIS AT RIVERSIDE STATIONS

Place.	On the river	Death-Rate per 1,000.	
Hyderabad ...	Indus ...	0·27	Average Death-Rate 2·23
Poona* ...	Mutamuli ...	1·43	
Lahore ...	Ravi ...	1·77	
Delhi ...	Jumna ...	1·78	
Kamptee* ...	Kanhan, Pench, Kolar	1·79	
Fort William...	Hugli ...	1·95	
Thayat Myo ...	Irrawadi ...	2·04	
Rangoon ...	„ ...	2·09	
Dum Dum ...	Hugli ...	2·14	
Allahabad* ...	Ganges, Jumna ...	2·18	
Jubbulpore ...	Narbada ...	2·81	
Benares ...	Ganges ...	3·81	
Barrackpore ...	Hugli ...	4·93	

learn the rates of mortality at stations situated considerably above the sea level, and as much as possible away from

its influence. With this view then the statistical results recorded at eighteen cantonments, of not lesser altitude than 1,000 feet, are here subjoined. For the sake of easy comparison they are subdivided into groups of varying heights, and besides the individual death-rates being placed in arithmetical order, the average death-rate of each separate section is given on the margin.

DEATH-RATES AMONG EUROPEANS FROM HEPATITIS AT VARIOUS ALTITUDES IN INDIA.

	Feet.	Mortality per 1000 strength.	Average Mortality.
(*a*) 1,056 *to* 1,200 *Feet Elevation.*			
Peshawar	1056	0·46	1·51
Nowshera	1200	1·57	
Lahore Fort	1130	1·77	
Meean Mir	1128	2·26	
(*b*) 1,476 *to* 1,500 *feet.*			
Neemuch	1476	1·88	1·83
Rawul Pindi	1500	1·55	
Nusseerabad	1500	2·36	
(*c*) 1,760 *to* 1,862 *feet.*			
Ahmadnagar	1830	0·65	1·87
Poona-Kirkee	1849	1·43	
Gwalior Fortress ...	1760	1·45	
Mhow	1862	1·58	
Secunderabad	1791	4·27	
(*d*) *Above* 2,000 *feet.*			
Satara	2320	0·59	1·36
Belgaum	2240	1·45	
Bangalore	3000	2·15	

(*e*) *Hill Stations.*

Chakrata	7056	0·98		
Cherato	7800	2·36	}	2·17
Darjeling	6941	3·17		

Here, again, are encountered most unlooked for discrepancies; for while at some of the cantonments of nearly the same altitudes—namely, of 1,130 and 1,200 feet, of 1,476 and 1,500 feet, of 1,760, 1,849, and 1,862 feet—the respective death-rates are tolerably uniform, at those of 1,128 and 1,130 feet, of 1,500 and 1,500 feet, of 1,760 and 1,791 feet, of 1,830 and 1,839 feet, of 2,240 and 2,320 feet, of 6,941 and 7,056 feet, very decided differences are found to exist. In the three following stations the disparity is strikingly remarkable, seeing that they are not only of about the same altitudes—the extreme difference being only 38 feet—and nearly in the same latitudes and longitudes, but have merely trifling differences of rainfall. Thus—

THE MORTALITY FROM HEPATITIS AT SIMILAR ALTITUDES, LATITUDES, AND LONGITUDES.

	N. Lat.	E. Long.	Feet Height.	Rainfall in inches.	Death-rate.
Secunderabad (St. John's Bell Tower) ...	17° 26′ 38″	78° 32′ 55″	1,791	27	4·27
Ahmadnagar	19° 5′ 55″	74° 46′ 57″	1,830	26	0·63
Poona (Kirkee)	18° 33′ 20″	73° 53′ 36″	1,849	44	1·43

I think all will agree with me that we have here a pathological climatic paradox well worthy the consideration of those having a taste for medical ratiocination. It is certainly one that I will gladly have solved for me. Its disentanglement is most assuredly puzzling, notwithstanding that we are all fully alive to the fact that latitude and climate are by no means twin sisters. From the circumstance that climate has quite as much to do with the topographical as it has to do with the geographical relationships of a place. From not only the position, but the nature of the soil, the altitude, the proximity of wood or water—running, or stagnant—moor or morass, and the direction of the prevailing winds, all playing an important part in the determination of those aërial influences which we denominate climate, therefore the climates of places on the same parallel of latitude and even in close proximity to each other, may be totally different It may possibly interest some to know that both Ahmadnagar and Secunderabad are situated on the table-land of the Dekhan, and are almost equally under the influence of the South-west Monsoon. The first-named station is, however, built on volcanic greenstone, and the last on a bed of granite. Their climates, however, are pretty much alike, both being cold till March, fine till June, and rainy till September. Facts which of themselves in no wise suffice to solve the mystery of the disparity of their death-rates. That of Secunderabad being $6\frac{1}{4}$ times greater than Ahmadnagar. So here, again, we need enlightenment. Meanwhile, in order to strengthen the above evidence that climate as considered by mere latitude and longitude has little, if any, controlling influence on the death-rate from hepatitis among the European residents in India. I herewith give the

DEATH-RATES AT TWO STATIONS IN ALMOST IDENTICAL LATITUDES AND LONGITUDES.

	Latitude.	Longitude.	Death-rate per 1,000 from Hepatitis.
Malliapuram ...	11° 3′ 2″	75° 51′ 21″	2·17
Calicut	11° 13′ 0″	75° 49′ 0″	4·40

These are assuredly, as Dr. Balfour who has given much attention to the question remarked to me, not alone most unexpected, but most unaccountable results. Indeed, the diversity in the whole of the death-rates in the table of altitudes is most perplexing, seeing that we not only know that as altitude above the sea level increases, temperature diminishes; but likewise that inflammatory diseases of the liver are proportionally less common in temperate than in tropical zones. Yet, notwithstanding this, we here find that the three lowest death-rates are met with at 1,056 feet (= 0·46), 2,320 feet (= 0·59), and 1,830 feet (= 0·65) respectively, and the three highest at 7,800 feet (= 2·36), 6,941 feet (= 3·17), and 1,791 feet (= 4·27). In what then, it may well be asked, consists the mysterious factor which so potently rules the mortality of Europeans from hepatitis in India, that even its naturally-supposed-to-be-healthy Hill stations are so deadly? As the Hill stations are of all others those which might have been *à priori* expected to be the most salubrious in regard to this disease.

Fearing as I do that if in India—the hot-bed of hepatic affections—cannot be discovered the *fons et origo* of their causation, the search is hopeless. I shall

attempt once more by statistics to discover a clue to the mystery which I think, the reader cannot have failed to see exists. Can it possibly have anything to do with rainfall? Let us see.

Relative Death-rate from Hepatitis at Rainy and Dry Stations.

The following statistics of nineteen stations have been kindly extracted for me from the Government Report by Dr. Balfour, and the only changes I have made is in the order of their arrangement, and in adding the calculated average death-rate to each table.

Those stations with an annual rainfall of under 45 inches are reckoned as dry. Those with an excess of 65 inches, as rainy.

Rainy Stations—Rainfall being above 65 Inches per Annum.

	Rainfall. Inches.	Ratio per 1,000 died from hepatitis.	
Calcutta	66·00	1·95	Average Death-rate 2·65.
Bombay	74·20	3·77	
Rangoon	99·69	2·09	
Port Blair	117·39	0·83	
Cannanore	121·60	4·11	

DRY STATIONS—RAINFALL BEING UNDER 45 INCHES PER ANNUM.

	Rainfall. Inches.	Ratio per 1,000 died.	
Debra Ismail Khan ...	7·00	2·01	Average Death-rate 1·44.
Multan	7·52	1·11	
Karachi	7·61	1·17	
Peshawar	12·00	0·46	
Bellary	17·33	1·28	
Hyderabad	21·00	0·27	
Lahore...	21·48	1·77	
Deesa	23·75	1.70	
Ahmadnagar	27·00	0·65	
Trichinopoly	31·00	4·84	
Belgaum	34 00	1·45	
Satara...	39·00	0·59	
Poona	44·00	1·43	

Here, apparently, we have something positive to go upon. Notwithstanding the discrepancies both in the rainy and in the dry stations being very marked, more particularly in the dry, where in one instance it is actually seventeen times greater at one place than at another although the difference in the rainfall of the two places (Hyderabad and Trichinopoly) only amounts in the

whole year to ten inches, as it happens that when the average mortality of all the stations is considered it gives us a death-rate of 1·44 in the dry, and of 2·65 in the wet, the idea is suggested that rainfall has possibly some influence on the mortality. Before making any further remarks on the value of this positive result, it is necessary for me to remind the reader that, although rainfall has an appreciable influence on atmospheric humidity, yet the actual rainfall itself furnishes in no instance an absolute estimate of the mean annual humidity of the atmosphere in any given locality. From the fact that the kind of soil, the nature of the vegetation as well as the topographical situation of the locality has a great deal more to do with the dryness or dampness of the atmosphere than the mere amount of rainfall. A low-lying flat, well wooded clay soil, even though seldom rained upon, may have an almost constantly damp supernatant atmosphere. While a high, sloping, gravelly, rocky, or chalky barren soil, with a far greater rainfall, may possess an almost constantly dry atmosphere. As the above table is on this account not altogether satisfactory, I herewith subjoin an additional one, which shows the death-rates of hepatitis in proportion to atmospheric humidity, irrespective of mere rainfall at 23 Indian stations :—

Minimum Atmospheric Humidity (*Under* 50°).

	Humidity.	Hepatitis Mortality	Average Death-Rate per 1,000
Multan	35°	1·11	
Rawul Pindi	40°	1·55	
Jubbulpore	44°	2·81	1·85
Debra Ismail Khan ...	44°	2·01	
Kampti	47°	1·79	

Medium Humidity (*between* 50° *and* 60°).

Deesa	51°	1·70	
Allahabad	53°	2·18	
Secunderabad ...	53°	4·27	
Hyderabad	54°	0·27	
Ahmadnagar	56°	0·65	1·72
Bellary	57°	1·28	
Karachi	58°	1·17	
Thayat Myo	58°	2·04	
Poona	59°	1·43	

Maximum Humidity (*above* 60°).

Lahore	61°	1·77	
Peshawar	61°	0·46	
Belgaum	63°	1·45	
Satara	69°	0·59	
Port William	74°	1·95	1·73
Bombay	75°	3·77	
Madras	75°	0·88	
Port Blair	75°	0·83	
Darjeling	84°	3·17	

It cannot fail to be here observed that, at individual stations there exists a still greater irregularity between humidity and death-rate than existed between rainfall and death-rate from hepatitis. For while the extremes in the rainfall table were 0·83 and 4·11, in this table of humidity they are 0·27 and 4·27. A noteworthy disparity.

Seeing that when the average of the death-rates are calculated in groups, according to the humidity being under 50°, between 50° and 60°, and above 60°. The relative proportions of mortality vary so little—from 1·72 to 1·85, it is impossible for the factor of humidity, alone to be regarded as either a specific predisposing or exciting cause of inflammation of the liver among Europeans residing in India. The death-rate is indeed seen to present no correspondence whatever with the atmospheric humidity. For it will be observed that even between 53° and 57° of humidity inclusive, we have actually variations in the death-rates of 0·27 and 4·27 per 1,000; and between 74° and 84° variations of 0·83 and 3·77. It is thus patent that some other factor, quite independent of atmospheric humidity, must be the determining agent of the mortality at the above named twenty-three stations. After making this remark, I daresay the reader will be not a little surprised to learn that, from personal observation I have arrived at the conclusion that a damp cold atmosphere is a most fruitful cause of all forms of congestive liver disease. This I explain on the principle that the influence of a cold, damp atmosphere upon the hepatic organ is indirect. Being exerted through the intermediary of its chill-favouring power. This idea is not only in harmony with the apparently respective influences of rain-fall and damp on the mortality from hepatitis shown in the

above tables; but coincides with the sea-coast, and riparian results; as well as with my experience of the effects of wet weather in favouring the attacks of all congestive forms of hepatic disease met with in England. For years past I have noticed that, just as every season has its fruits and flowers, and every season its crops of diseases, as regularly as the fall, and budding of the leaf comes round a larger percentage than usual of congestive liver affections fall to my share of treatment. And what is more to the point, many of the same cases of hereditary, as well as of acquired liver affections walk into my room every spring and autumn. This cyclial return of patients has convinced me that the mere factor of cold damp is a notable exciting cause of hepatic congestions, and is due to its proclivity to engender chills. For almost all such patients blame the weather as the cause of their attacks. Particularly those of them who have at one time resided in the tropics, either of Asia, Africa, or America, and thereby become more susceptible to take cold in this country during the changeable spring and autumn months of the year. This idea, it will be noticed, though entirely in accord with that part of Dr. Moore's theory of Indian hepatitis being excited by chills, in no way favours the assumption, he has superadded to it, and that too as a *sine quâ non*, of there existing a special liver-chill producing influence in Hindustan—differing from what exists in, and is ever at work in all other quarter of the globe.

Deductions regarding the Cause of Hepatic Abscess, and its Antecedent Inflammation of the Liver, from the Adduced Data.

A deduction being little else than a theory, and to propound a theory being easier than to ad-

duce logical reasons in its support. The point at which I have now arrived, I approach with considerable diffidence. For, if, as we know, in order to be able to interpret nature's secrets aright one must have both coinciding and positive facts to deal with, it can in no case be an easy matter to elicit correct opinions on the etiology of morbid phenomena when the data at his disposal for the disentanglement of the problem do not, as in the present instance, possess the advantage of being either definite or uniform. It is generally considered indeed, to be the soundest policy when the facts are neither numerous enough, positive enough, nor sufficiently in concord to admit of inferences being drawn from them, consonant alike with experience and reason, to delay theorising until the scope of one's mental vision becomes more extended by the contemplation of better data. This, no doubt, is philosophic wisdom. But, alas! it is totally inapplicable in the present case. For as medicine, as a science, is not only still in its infantile cradle, but even not so much as yet freed from its swaddling clothes, she absolutely requires the helping hand of theory for her emendation.

Theory being the mortar which cements together the knowledge already possessed into the stepping stones towards what we are in search of. In no advancing art, science, or philosophy can it be entirely dispensed with. While in medical science, which is at this moment a rapidly progressing one, it is an absolute essential to its advancement. Were we, indeed, foolish enough to try and dispense with the aid of theory, until every link in the chain of rational medicine is perfected, all progress would be immediately arrested, and the dawn of exactitude still further delayed.

Theory must, however, be always regarded in its own

proper light. Not as a permanent; but merely as a temporary means to an end. It must be looked upon exactly in the same way as is the temporary scaffolding employed by the architect—which he gradually raises as his building advances and after it has served its turn casts aside. For theory being but the mental scaffolding of knowledge is equally ephemeral in its nature as is the physical scaffolding of the builder. And like it too, after having served its turn may be unhesitatingly cast aside. Nevertheless, even although aware that the theory which we regard as true to-day, may be found to be false to-morrow, it ought not only to be appreciated, but fostered and cherished so long as its career of usefulness lasts. Moreover, as it is solely by the acquisition of new data that we learn, and the more we learn, the more our ideas expand, no one need be ashamed of changing his opinions in an advancing science. On the contrary, he should feel proud of being the possessor of a mind sufficiently susceptible and liberal, to change them in proportion as his knowledge advances. For though his facts may be as everlasting as they are indisputable, his theories and opinions must ever be as apocryphal as they are ephemeral. Such being the case, although I have dealt boldly and decisively with the foregoing figures and facts, I must be pardoned if I advance the theories I am about to propound with caution and hesitation. And seeing that not alone is their birth the product of necessity, but that the nature of the subject demands that I should launch them, such as they are, on the ever ebbing and flowing sea of medical opinion, I trust my professional brethren, no matter what their phases of thought may be, will carefully reflect upon the pros and cons of the whole matter, ere they pronounce judgment. For all know that as every

question has two sides to it hasty generalisation is rarely the parent of wise deduction.

From its unfortunately happening that when one is dealing with such an abstruse problem as the causation of disease, the difficulty of the task is in no way diminished by the expounder having a limited space at his disposal; in order to enable the reader at a glance to grasp the intrinsic value of the figures and facts in the tables upon which I ground my deductions, and aid him in arriving at the inevitable conclusion to which, I think, they point, I shall give the following short epitome of the most salient and noteworthy data the tables appear to me to contain.

Firstly: The statistics not only clearly show that, for some reason or another, the mortality among Europeans from inflammation of the liver, whilst resident in India, increases with the period of residence, and that it is quite different in the three Presidencies of Bombay, Bengal, and Madras; but that it varies in an extraordinary manner in different localities. For, as will have been noticed, the sea-coast, river-side, inland, high, hilly, dry, and rainy stations over the whole of British India show great inequalities in their death-rates; varying, indeed, from 1 to 17 times more at one place than another. The lesser variation being in stations of from 1,476 to 1,500 feet high, of similar longitudes and latitudes, and of minimum atmospheric humidity. The greater variations being, on the other hand, among those by river-sides of medium atmospheric humidity, and minimum rainfall.

Secondly: The mean death-rate of each group of stations varies only 1·94 per cent.

Thirdly: The six highest death-rates are met with at the river-side stations on the east of Hindustan (4·93);

the dry, in the south (4·84), the sea-coast, on the west (4·40), the high inland (4·27), and the rainy (4·11). Whilst the lowest death-rates, strange to say, are likewise met with at the river-side (0·27), the high inland (0·46), and the sea-coast (0·83) stations.

Theory regarding the Causation of Inflammation and Abscess of the Liver Deduced from the foregoing Hospital Statistics.

We have now arrived at a point of much importance to us as practitioners of medicine. For, as an effect ceases so soon as its cause is removed, until we know what the cause of a disease is, it is next to impossible for us to expect to be able to remove it. Knowing the cause, however, we may possibly have much in our power. The statistics adduced have taught us the following facts:—

1st. The greatest average mortality is at rainy stations.

2nd. The highest, as well as the lowest, of all the death-rates is at riverside stations.

3rd. Places at identical altitudes have different rates of mortality.

4th. The death-rates at places in the same latitude and longitude have no correspondence whatever to each other.

At first sight these, and the other facts revealed by the tables appear so contradictory and perplexing that one might almost feel tempted to cast their consideration aside in despair. A little reflection, however, shows that, paradoxical although it may seem, the information yielded is so very decisive on one particular point as to

be of the utmost value to us as investigators of the etiology of disease. For all the data, singly as well as collectively, point in no ambiguous language to the fact that the mere factor of LOCALITY—whatever that may be—is a far more powerful element in the production of hepatitis, and consequently of abscess of the liver in British India than all the other numerous exciting causes either separately or combined—a piece of information which opens out to the sanitary-trained mind an entirely new vista of thought. For it proves that there exists some—as yet unknown—controlling exciting power over the mortality from hepatitis totally independent of mere geographical situation, and its concomitant climatic influences. This becomes tremendously apparent when it is shown that places in close proximity to each other have entirely different death-rates. Thus, three military stations on the river Hugli are so near to each other that, as the crow flies, the distance between them is less in the one instance than five, and in the other than ten, miles. And, notwithstanding that at each of them the troops are treated, as regards housing, feeding, clothing, and exercising, in precisely the same way the death-rates from hepatitis among them are notably different, being at—

Fort William	.. ..	1·95 per 1,000.
Dum Dum	.. .. ..	2·14 „
Barrackpore	.. .. ..	4·93 „

Startling as this discrepancy is, it is yet capped by another—namely, that while Madras on the sea-shore, and St. Thomas Mount, within five miles of the coast, are only eight miles apart, the death-rate at Madras is as low as 0·88, while at St. Thomas Mount it actually reaches as high as 4·67—that is to say, it is 500 per cent.

more. And this, too, in spite of the troops at Madras living within the walls of a fortress, and those at St. Thomas Mount in open cantonments, which might not unreasonably be supposed to be more healthy. But this is not all. Still another fact connected with St. Thomas Mount is well worth recording, and that is, that in 1836 a large draft of young recruits arrived from England, some for the horse, some for the foot artillery, and although soon after their arrival several cases of hepatic abscess appeared among the cavalry, not one single case occurred among the infantry. And yet the barracks of the two were only half a mile apart, the horse soldiers being stationed on the south-west, the foot soldiers on the east side of the cantonment, and both being in all other respects apparently under exactly the same influences. Dr. Balfour, who was serving under Surgeon Geddes at the time, says that, although they had many conversations on the subject, they could not even frame a guess at the cause of the one set of recruits being affected and the other not. The only thing they could think of was the riding exercise. But that, of course, is an untenable view, for if riding were a cause of liver abscess we would have abundance of them in every mounted regiment. Here, then, is a problem requiring solution.

No thinking man can help forming a theory on this point, and the following is the one I have formed:— Believing as I do that not alone every great, but equally every small, event taking place on this earth of ours, as well as in the universe at large, is governed and regulated by definite, immutable, and universally-applicable laws. "All seeming discord in Nature must be but harmony not understood." Therefore, although the contemplation of the figures in the foregoing tables is undoubtedly at first bewildering, I think if we construe them aright, and

deduce the unknown from the already known, it is not improbable that out of their seeming discord may be evolved philosophic harmony.

The figures in the tables having incontestably demonstrated that climatology, in the ordinary acceptance of the term, is not the ruling factor of the causation of hepatitis, and equally clearly shown that there are permanent foci of the disease in circumscribed localities scattered over the length and breadth of British India, the question forced upon us is—Wherein consists the local disease-producing agency? That is to say, in what element or group of elements dwells the mysterious influence of locality in exciting or in predisposing the liver to become inflamed? Does the "cause" lie in the soil, exist in the water, float in the air, or pervade the natural and artificial surroundings? Its marked limitation to particular localities entirely excludes the idea of its being due to human non-sanitary agencies. For precisely similar human non-sanitary agencies are more or less equally at work in all Indian cantonments. Whence, then, it may well be asked, springs the deleterious agent? No one can, of course, answer in the present imperfect state of our knowledge a single one of those questions decisively; but many among us may probably guess at the nature of this disease-producing agent, and imagine it to be a parasite. This is, at least, the view I feel inclined to take of the question, and for the following reasons:—

Every biologist knows that a parasite, no matter whether it be small or large—no bigger than a microscopic cytoblast, an undifferentiated piece of protoplasm, amœba, or an organised thirty feet long tapeworm—forms an integral portion of the earth's fauna quite as much, in the eyes of the philosopher, as an elephant or a

gigantic sequoia sempervirens. (*a*) All are equally aware that not only are the habitats of the elephant and the sequoia exceedingly localised, but that indigenous plants and animals of all kinds have likewise, as a rule, a very circumscribed area for the development and propagation of their species. So circumscribed, indeed, is it in the case of some plants that, while a certain species may be found growing and flourishing abundantly on one side of a hill, not a trace of it may exist on the other. Nor may even so much as a single individual of the same species be again encountered for miles and miles round. What holds good for plants—which are, in a measure, fixtures—holds equally good for many animals that are devoid of the means of free locomotion. No better example of this can I give than by citing the case of the Italian edible snails, which were imported into Britain by the Roman soldiers 2,000 years ago. To this very day the descendants of these same snails are still to be found, and only to be found, in the localities in England in which they were originally cultivated by their military importers. And notwithstanding that countless generations of their descendants must have successively passed away since then, the race still flourishes.

Considering it then, as settled that the natural fauna of a country is always limited to circumscribed localities, I shall proceed to show to what portion of the earth's

(*a*) While in California during my autumn tour in 1884, I visited the Mariposa Grove, one of the limited localities where these trees grow, and I drove in a carriage and four through a tunnel of 10½ feet wide, and 12 feet high, made in the living trunk of one of them. The circumference of the tree is 102 feet; its computed height 400 feet; a portion of the bark which I measured was 22 inches thick; and a 14 inch section of a piece of the wood, which I have had made into a ruler, contains 210 rings. So if each ring represents a year's growth, the tree is over 3,000 years old.

fauna I attribute the production of hepatitis. This I can answer in one single word—Germs. Yes, disease germs, insignificant though they be, have in reality an equal right to be considered a portion of the earth's fauna, as the elephant or the stupendous sequoia, and may, like them too, possess very circumscribed indigenous habitats, where they live, propagate their species, flourish, and die. This being once admitted, there is no difficulty in understanding why certain localities in British India should be hotbeds of hepatitis, while others in close proximity to them are much less affected with the disease, and, again, others at a distance seventeen times more so. For the parallelism in great and small is perfect.

This germ theory of hepatitis and suppuration of the liver I have already called the attention of the profession to, and a very little reflection will show it to be well founded. For germs being parasites, and the liver being the organ in the body most liable to be affected with a variety of both animal and vegetable parasites—such as hydatids, flukes, filaria, and a hyphomycetous fungus allied to the Actinomyces bovis—there is no difficulty in the way of believing in the possibility of germs being one of the exciting causes of hepatic abscess and its antecedent inflammation of the liver. In fact, the more the pathology of hepatic diseases is studied, the more evident does it become that parasites—vegetable as well as animal—play an important part in their production. And once the idea of germ action is admitted as a possibility in the production of hepatitis in India and elsewhere, the whole mystery of LOCALITY melts into thin air. And not only so, but all the other apparently anomalous facts brought to light by the afore-cited statistics blend themselves into harmonious concord, instead of remaining apart in disunited antagonism. Even the fluctuations in the death-

rates from hepatitis and abscess of the liver, which are noticed at the different stations, are readily accounted for. For just as there are irregular periods of accressence and decressence in the life history of all animated nature—particularly noticeable in the good and bad seasons of fruits and flowers—so in like manner, no doubt, there are fluctuating periods of accressence and decressence in the life-career of those minute disease organisms which we call germs, be they animal (epizootic) or vegetable (miasmatic). This is made patent by the fact that great waves of parasitic epidemics, in the form of spores, mycelii, and fungi—ever and anon sweep over the vegetable world, recent striking instances of which we have had in the blight of the potato and vine plants. And what holds good for the vegetable world holds equally good for the human species, as we know by our epidemic diseases, whether the parasite attacking it be a vegetable or an animal; or its finding entrance into the body in the shape of a spore, a mycelium, germ-spawn, or even as a disease-protoplasm. Once within the liver the germ finds an appropriate nidus for its wants, soon propagates its species, founds a colony, and, by acting like any other foreign body, induces an effort of nature to cast it out in the form of inflammation and suppuration of the surrounding tissues. As some of my already published views have a most important bearing on this question, and I have not sufficient time at my disposal to repeat them, I take the liberty of referring the reader to what I have written regarding the etiology of the periodicity of contagious and infectious diseases (*a*), contagious jaundice, malarial hepatitis, and paroxysmal congestive hepatic

(*a*) Chapter ii. of an essay entitled "Some new Facts connected with the Action, of Germs in the Production of Human Diseases."—*Medical Times*, 1881.

hæmaturia, all the mysterious characteristic, though anomalous, symptoms of which may be readily explained on the theory of its being due to disease germ action (*a*).

As further evidence in support of this germ theory, I may remind the reader that it was abundantly shown by the adduced statistical tables that the highest rates of mortality were in all cases met with in localities exposed to aqueous influences—to wit, rainy, river, and sea-side stations—at the same time as they conclusively proved that a more potent factor than moisture was at work in producing the disease. Two facts which, when taken together, show that the influence of moisture is indirect, and that its influence on the disease in all probability is through the intervention of a living organism. This is rendered all the more probable when it is remembered that moisture is essential to the growth of all forms of epizootic as well as miasmatic germs, and that all kinds of known fungoid growths flourish most abundantly under the influence of heat and moisture. There exists, therefore, no difficulty in accounting for damp situations being the most fertile in the production of hepatitis and abscess of the liver on the germ theory. But, of course, there are also other exciting causes at work there as elsewhere. Among the exciting causes of abscess of the liver met with in all climates are the direct effects of cold and wet, biliary concretions impacted in the bile ducts, inflamed and dead hydatids in the tissue of the liver, as well as embolisms in its blood-vessels. But suppurations in the nitestinal tract are of all others the most frequent cause of hepatic abscess, no matter what the suppuration may be due to. Dysentery, a stricture of the rectum, a pin or a fish-bone in the appendix vermiformis. Even a purulent

(*a*) "Diseases of the Liver," Under the headings of Germ Action.

absorption from a gonorrhœa, has induced an abscess in the liver. Direct injury to the organ is likewise an exciting cause of abcess.

The Relative Frequency of Abscess of the Liver in England and India.

In the first chapter of this essay I promised to show that abscess of the liver is as common a disease among us as it is among Europeans residing in the tropics. I shall now fulfil that promise by placing before the reader hospital statistics which prove it.

Brigade-Surgeons H. Cook and Vandyke Carter, and Surgeon-Major Hojel, furnished Dr. Moore with the following statistics of liver abscess occurring in Bombay during the past three years :—

	Number of Patients.	Per cent. having Abscesses.
In the Jamsetji Hospital . .	18,759	0·62
„ Goculdas do. . .	7,397	0·54
„ European General do.	4,235	0·66

These figures show that out of a total of 26,156 native patients treated in the Bombay hospitals, 0·58 per cent. laboured under hepatic abscess ; and of 4,235 Europeans, 0·66 per cent., that is to say, only ·08 per cent. more; while Dr. Norman Moore gives statistics of 2,464 autopsies at St. Bartholomew's Hospital, London, in 20 of which abscess of the liver was met with, thus showing that no less than 0·81 per cent. of Europeans dying in temperate England suffer from abscess of the liver in one form or another—that is to say, 0·23 per cent. more than the natives of India, and 0·15 per cent. more than Englishmen residing in India ! No wonder, then, that I said that

suppuration of the liver was a far more common disease in England than was generally supposed. I am not now speaking of any particular variety of abscess, but of hepatic abscesses in general; for all kinds of abscesses are met with in India, as well as in England, and no distinction that I can see is made in the above-cited Indian hospital statistics.

The Diagnosis of Inflammatory Affections of the Liver.

I shall now proceed to place before the reader what I consider to be the most salient factors conducive to the proper diagnosing of any given case of inflammation of the liver.

Signs and Symptoms of Hepatitis.

It being chiefly on a recognisable increase in the size of the hepatic organ, upon which we depend in estimating the extent, as well as the gravity of an inflammation attacking it, I may begin my remarks on diagnosis by reminding the reader that although the actual cubic dimensions of the liver is much about the same in all persons of similar ages, the normal dull area varies not only in a man and woman, but according to the height of the individual. In a person of 5 feet 7 inches it is calculated at 4 inches in the perpendicular right nipple line. In one of 5 feet at $3\frac{1}{2}$ inches. While in one of 6 feet or more $4\frac{1}{2}$ inches are thought to be about the average extent of the dulness. The liver's dulness is usually said to begin about two inches below the nipple. But where it

begins, or where it ends, is of comparatively little consequence; from the fact that some people have naturally very long suspensory ligaments, and in these the liver is much lower in the abdomen. While in women its position is often considerably altered, merely on account of their mode of dressing. Consequently, the actual extent of the dulness, and not the position of the dulness, is what is to be regarded in estimating the volume of the organ. The left lateral margin in most persons ends at the lower left edge of the xiphoid cartilage.

In percussing a patient's liver, one must always guard against making a mistake in calculating the dulness, if there be either fluid in the abdomen, gas in the stomach, or an accumulation of fæces in the transverse colon. As well as the risk he runs of confounding the dull note elicited from a hydronephrosis, ovarian tumour, or even an enlarged spleen. All of which conditions have repeatedly led to errors in diagnosis. It has happened too that even in cases of right pneumonia and pleurisy the dull percussion note has been so distinctly continuous with that of the liver as to lead to their having been compounded together.

The general symptoms of hepatitis are:—A sense of fulness and discomfort in the hepatic region. In some cases amounting to actual pain on deep inspiration and when lying on the right side. Strange to say some patients can lie on the right and not on the left side, on account of what they describe as a feeling of dragging. Tenderness on percussion, and acute pain on firm pressure. More or less increase of dulness, especially in the perpendicular right nipple line. Instead of the dull extent being normal, it may be as much as six, eight, or even ten or more inches. A hot and dry skin, with pyrexia. Sallowness of the skin, sometimes amounting to

actual jaundice. Yellow conjunctivæ, foul tongue, occasionally offensive breath, and rapid pulse. Urine scanty, high coloured, and, on cooling, turbid, with a great deposit of lithates—pink, ochre, or red coloured—sometimes albuminous, but always (except when kidney disease also exists) of a specific gravity of over 1012. The bowels are generally constipated, and if there be jaundice there are light or pipeclay-coloured stools. If these symptoms are present, there need be no hesitation felt in diagnosing the case as one of acute inflammation of the liver. Although in all cases of hepatitis the complexion is more or less sallow, it is only when the *whole* of the liver is inflamed that there is actual jaundice. The jaundice being the result of suppression of the hepatic functions. For just as we have suppression of the urine, from the kidneys striking work in cases of nephritis. So we have jaundice, from the liver striking work, in cases of hepatitis.

In cases of chronic hepatitis hæmorrhages, in the form of epistaxis, hæmatemesis, bloody diarrhœa, copious bleeding from piles, and hæmaturia, as well as menorrhagia in the case of females, are not uncommon. But the worst complication of all is ascites.

Inflammations of the liver have been sub-divided into many varieties. However, I shall only allude to the more important of them.

One is called peri-hepatitis, from its being supposed that the capsule surrounding the liver is the chief seat of the inflammation. It is recognised by there being a distinct friction sound audible in the right hypochondriac region, and from it being only heard during the respiratory act, it is said to be frequently mistaken for the friction sound of pleurisy.

Another variety is named sympathetic hepatitis. It is usually the concomitant of pneumonia, and is believed to

be dependent upon an extension of the inflammation of the lung to the liver, through the intervention of the sympathetic and pneumogastric nerves. In the same way as inflammation of one eye is communicated to the other by direct nerve influence.

A third variety is entitled Malarial Hepatitis. And this is the most important of all, as it includes within its capacious boundaries all forms of inflammation of the liver arising from one severe, or many repeated slight attacks of ague and malarial jungle fevers. It is a form of disease from which the system seems scarcely ever able to free itself. Like syphilis, the malarial poison appears to saturate the system, and render the patient, at any future period of his life, liable to its influence. A few years ago I saw, along with Mr. Phillips, of Leinster Square, an Indian officer, æt. 70, threatened with hepatic suppuration, with distinct aguish symptoms. Notwithstanding that it was twenty years since he had returned from India; where he had suffered not only from jungle fever, and true aguish attacks, but from dysentery in its worst form.

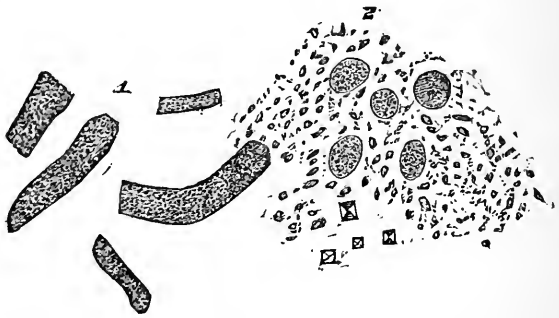
Malarial liver disease is often accompanied by paroxysmal hæmaturia. The main features of which, as I pointed out, many years ago (*a*) is that while the urine is bloody in appearance it yet contains few if any corpuscles. Nothing but their *débris*—granular tube casts and oxalate of lime crystals, and large granular cells, as shown in the wood cut.

A fourth variety is known as syphilitic hepatitis. This, I think, ought scarcely to be called a specific form of disease; for it is not, I believe, due to the poison of syphilis, but is ordinary hepatitis, occurring, perhaps, in an aggra-

(*a*) Royal Med. Chir. Soc. Transactions, 1865

vated form in a syphilitic constitution. It has no relationship whatever to, although it may be coincident with, what is known as "syphilitic liver," which is a constitutional affection due to syphilitic poison, whose chief pathological characteristic is the deposition in the liver parenchyma of well-defined circumscribed gummatous nodules, varying in size from small microscopic objects up to the dimensions of an orange. These deposits have nothing in common with hepatitis. Being merely local manifestations of a constitutional disease action. In fact, a tertiary form

FIG. 1.



1. Granular tube-casts.
2. Large granular cells.
5. Oxalate-of-lime crystals.
6. Amorphous urates.

of syphilis, always associated with its cachexia, and usually unattended by any inflammatory disease action.

In the so-called syphilitic hepatitis the enlargement is sometimes so considerable, and the tissues so hard, that it becomes exceedingly difficult to determine whether or no suppuration is about to take place. While in others the left lobe is so bulging as to give rise to the suspicion of hydatid disease. An interesting case of this kind, which I saw a year ago with Dr. Macpherson, I will briefly relate, as I think it shows how sometimes unsuccessful exploratory puncturing of the hepatic tissues may not only produce no bad effects, but possibly, lead

to a salutory result on an enlarged and indurated liver. I punctured the liver of a gentleman, æt. about 35, in 3 places on the 13th June, and on the 20th December I received from Dr. Macpherson a letter containing the following remarks regarding the case:—"The punctures were not followed by any irritation: but the patient remained low and feverish. After a few days I allowed him to visit his friends in Ireland, where he rapidly improved under the influence of iodide of potassium and bichloride. But the improvement was no doubt materially assisted by sea air and out-door exercise. Having improved so far I sent him to Aix-la-Chapelle to undergo the inunction cure. This was carried to salivation. In six weeks he returned well and in high spirits. I found the liver diminished in size, but the left lobe was still enlarged, with a thick gummatous edge. As he said he felt perfectly well I sanctioned his returning to India, which he did in a couple of weeks.

It will be noticed that Dr. Macpherson makes no remark regarding the salutary effects the punctures had on the case. This arose from his failing to appreciate it. But as the case was not improving before the liver was punctured, and it began to improve immediately afterwards, and continued steadily to improve. I have no doubt in my own mind—especially from my familiarity with the history of similar cases—that the puncturing of the organ was the turning point of the disease.

The General Treatment of Hepatitis.

In a case of acute hepatic congestion the first thing is to enjoin strict rest. The second is to put the patient on low diet. The third to freely clear out the bowels. The fourth to

relieve the local discomfort by the application of hot, thick, and large linseed poultices. The fifth, if there be signs of threatening suppuration, to apply a freezing mixture of ice and salt, or leeches, or even cupping-glasses over the painful hepatic region. It is sometimes surprising how speedy and complete is the relief afforded by this indirect mode of local depletion; but I shall presently allude to a much better method of relieving an engorged liver, to which I have given the name of hepatic phlebotomy, from its being in reality a mode of bleeding the liver itself. Euonymin, iridin, leptandrin, podophyllin, and all other forms of hepatic purgative stimulants, are totally inadmissible in cases of hepatic congestion. Mercury is here our sheet-anchor, both as a purgative and an antiphlogistic.

When hepatitis, no matter what its variety may be, has once reached the congested indurated stage of so-called hypertrophic cirrhosis, I have found much benefit to accrue from:

Puncturing the Capsule of Glisson.

So soon as the liver of a patient is not only enlarged, but hardened, in consequence of the pressure to which its inflamed tissues are subjected, as the result of their engorgement and confinement within their inelastic fibrous covering. I know of no more effectual way of giving relief than by puncturing the capsule of Glisson. I was led to adopt this plan of treatment from my personal experience of the benefits derived from puncturing the distended sheath of the sciatic nerve in cases of acute neuritis, which is done on precisely the same principles as the surgeon punctures

the tense unyielding tunica albuginea to relieve pressure on the inflamed secreting structures of the testicle in cases of orchitis.

The operation of puncturing the capsule of Glisson is done with a trocar of the diameter of a No. 2 or 6 catheter, according to the gravity of the case. And as there is in this, as in every other surgical operation, a wrong as well as a right way of performing it, I will give an example showing how simple and safe it may be when properly done. The case I shall relate is not only a recent, but, I may add, the most successful one I have as yet had.

The patient (a Dane by birth), æt. 52, first came to me in July last from Rio Janeiro, with a liver enlarged and indurated in all directions. In the perpendicular right nipple line it measured over eight inches. He had suffered from jaundice two years before. No doubt the result of suppression from a general hepatitis, the *fons et origo* of which was admittedly free living in a hot climate. Under the use of mercurials and alkaline salines he rapidly improved, and continued his journey to Copenhagen. I saw no more of him till the 15th Oct., when he returned to London greatly recruited in general health. He had been for six weeks at Carlsbad under Dr. Kraus. The liver was, however, still enlarged and indurated. The left lobe extended about two inches beyond the xiphoid cartilage; but the right was materially diminished in size. For it now measured only six inches in the right nipple perpendicular line. From his being anxious to return to South America as speedily as possible, I proposed to puncture his liver's capsule in three different places. To this he readily assented, and on the 18th I punctured the left lobe with a No. 2 trocar in two places, and the right lobe with a No. 5 in one place, keeping the instruments in the wounds for several minutes. He had no anæs-

thetic. I left him at the Langham Hotel, where he was staying without any nurse, simply giving the chambermaid instructions how to apply hot poultices if necessary. And what was the result? On visiting him at 9.30 next morning, that is to say, exactly eighteen hours after the operation, I found he had breakfasted, and was then busy at his table writing letters, with not a single bad symptom. All he complained of was tenderness at the seats of puncture. In three days after the operation he walked to my house, a distance of five hundred yards, saying he felt quite well. On examining the liver, to my astonishment I found it had greatly diminished in size, three-quarters of an inch at least, towards the left side, and all tenderness on pressure had gone. On the 6th Nov., that is to say, exactly three weeks to a day after the operation, he called to say good-bye, as he was to start next day for Rio Janeiro. And he might almost be said to be a cured man, in as far at least as the dimensions of the liver were concerned. For it measured now scarcely five inches in the perpendicular nipple line, and the left lobe only extended half an inch beyond the xiphoid cartilage. Be it remembered I do not cite this as an average case. For on the contrary, as already said, I never had such a successful one before. My object in citing it, therefore, is only to show what may be done under favourable circumstances.

From this, the treatment of congestive induration by puncture, I pass to something still more interesting. It is the treatment of acute hepatitis by direct

Hepatic Phlebotomy in Inflammation of the Liver.

This title sounds like a novelty. And no doubt a novelty it is. But everything must have a beginning,

and although no one has as yet, as far as I am aware, except myself, ventured to withdraw blood directly from a human liver, I have but little doubt but that there must have been hundreds of people who, like myself, have revolved this subject in their minds, from having marvelled and marvelled in vain for a rational explanation of the why and the wherefore we leech and cup the abdominal parietes in cases of acute hepatitis. From the fact that the parietal blood-vessels of the abdomen have no direct connection whatever with those of the liver, consequently the withdrawal of blood from them can not have the slightest influence upon the amount of blood in the hepatic organ, except in so far as it diminishes the total amount of blood in the patient's body. This is a palpable truism, seeing that the only channels by which blood is conveyed to the liver are the hepatic artery, a branch of the cœliac axis, and the vena portæ, formed by the union of the veins of the chylopoietic viscera. Both of which sources of blood supply enter the gland by the transverse fissure. While, on the other hand, the channel of the blood's exit from the liver is solely the hepatic veins, which pass out at the posterior border of the organ, and empty themselves directly into the vena cava. It being thus seen that none of the hepatic blood-vessels communicate with those of the abdominal parietes, it is evident that it must be utterly impossible that the withdrawal of blood from the vessels in the abdominal parietes can have any direct effect upon the quantity of blood in the liver. Well, then, it may be asked, upon what principles do we either cup or leech the abdominal parietes in cases of acute hepatitis? Common sense makes no reply.

Hepatic Phlebotomy.

It is usually accepted as a proverb that "fools rush in where angels fear to tread." But like most other wise sayings, this one, too, has its antithesis. For the equally wise saying that "knowledge inspires confidence" stands in exact antiposition to it. Though not a surgeon, or making any pretence to be one, I think I may with something like justice opine that ten years' practice as an experimental physiologist taught me many things that no surgeon would be likely to learn in the field of human practice. And one of these was that a certain amount of blood can be directly withdrawn from the livers of dogs, not only without danger to life, but without any apparent inconvenience to the animal. And further, that on opening the abdomens of the animals within a few days afterwards it is scarcely possible to detect the seat of the hepatic punctures. And that even when they are detected, there is no inflammation around them. Nothing but a small red spot is in general visible on the capsule of the liver. This observation I first made at the time I was working at the artificial production of diabetes in animals by the injection of stimulants into the portal vein.(*a*) And although during years afterwards I had often thought of the possibility of performing with advantage hepatic phlebotomy on the human subject, I never had the courage to propose it. Until I saw how many poor patients succumbed to acute hepatitis, where it appeared to me the direct withdrawal of blood from the liver might possibly have arrested the

(*a*) "Recherches sur la Physiologie du Diabète Sucré." *Compt. Rend.* Soc Biol. V., pp. 59-61. Referred to in the writer's work on "The Urine and its Derangements," p. 212.

progress of the case, and saved the life of the sufferer. Accordingly I gradually began, in suitable cases, to propose the performance of the operation. But time after time was I baffled. Sometimes by the patient alone, sometimes by both patient and doctor, so soon as they learned that the operation was a new and untried one, and the only experience I had of it was on dogs. At length a case came before me so palpably favourable for the operation, and so patently hopeless without it, that I urged its performance, and hoped I had gained my point. But in spite of my telling both patient and friends that my experience in such cases was that four out of every five died; indeed, that I had scarcely ever seen any person in his then state recover, the chance offered him by hepatic phlebotomy was nevertheless declined. The case was that of a strongly-built man of thirty years of age (who I saw along with Mr. C. L. Evershed in Sussex), who had for several years been addicted to intemperate habits, but who had, notwithstanding his habits of free living enjoyed comparatively good health until he was attacked with hepatitis within three months of my being called to him. When I saw him the liver was much enlarged, and very tender on pressure. It was impossible to ascertain its exact dimension from the abdomen being greatly distended with ascitic fluid. The lower extremities were œdematous; his pulse was quick, his temperature high, and his respiration rapid. He appeared to me to be in a hopeless state; so I strongly advised the performance of hepatic phlebotomy. The result is told in the following letter from Mr. Evershed:

"Our patient died just a fortnight after you were here. I tapped the abdomen three times; but it filled again I could not get the consent of the friends to puncture the liver although I tried hard to do so. He became

comatose in the middle of the day, and died during the night."

Such being about the usual history of these cases there is no wonder that I was so desirous for the performance of an operation which appeared to me to afford the only chance of hope. Moreover, as the patient was both young and strong, and his dying was apparently entirely due to the intensity of the inflammation, and feeling that in all human probability the relieving the conjestion of the liver by direct depletion would have saved his life, I determined in the next suitable case not only to urge the performance of hepatic phlebotomy; but to offer to do it myself. And so I did. As there is nothing so convincing of the value of one's opinions, as the fact of their leading to practical success, I shall now relate the history of the next suitable case in which I was consulted. It happened about five months after this, and I am glad to say had a diametrically opposite result.

Along with Dr. Dunbar Walker I saw a married lady, æt. 38, who might be said to be of intemperate habits. A month before our consultation she was attacked with hepatitis, and when Dr. Walker was first called in he "found the liver (I extract this from his history of the case) already greatly enlarged and indurated. The legs œdematous, and the abdomen contained a large quantity of fluid. The abdomen was tapped, and aperients given. As these remedies were persevered in for a fortnight without a diminution in the size of the liver, or any permanent improvement in the general symptoms taking place, Dr. George Harley was called in, and recommended bleeding the liver by a trocar." Now I shall myself take up the thread of the narrative. This case was the exact counterpart of Mr. Evershed's, except that the patient was aged 38 instead of 30, and a woman instead of a

man. Hope of saving her by the ordinary routine of orthodox practice I had none, consequently I urged even more emphatically than I had done in Mr. Evershed's case, the propriety of giving the patient the chance of a forlorn hope by hepatic phlebotomy. It was declined by both the patient and her husband; on reflection, however, after the utter hoplessness of the case had been forcibly put before them by Dr. Walker, who strongly supported me, their mutual assent to the operation was obtained, and on the 4th of October, 1883, was performed for the first time, as far as I am aware, the operation of hepatic phlebotomy.

Dr. Walker having rendered the patient insensible with the A.C.E. mixture, I pierced the liver from right to left, with an eight-inch long trocar, of the diameter of a No. 3 catheter. The normal liver being at least ten inches broad in an adult woman, and it being greatly enlarged in this case, I knew I was perfectly safe, and so ran the trocar up to its very hilt, in the hope that during its penetration it would wound some vessel of sufficient calibre to yield a free stream of blood. All now had to be left to the chapter of accidents as regards the risk of the point of the instrument lodging in the interior of a blood vessel and allowing the entrance of air.

It being impossible to overlook this important risk, I had previously gone carefully over hepatic literature in order to discover if there was any example on record where such an untoward accident had occurred, either during the exploration of the liver or in the tapping of abscesses, or hydatids of the organ with trocars. I found none. But I came on a case reported in the eleventh volume of the Clinical Society's Transactions, p. 230, under the title "A Case of Sudden Death following the Operation of Tapping a Hydatid Cyst of the Liver," and I be-

lieve erroneously given as an example of death from the entrance of hydatid fluid into the trunk of the portal vein: My opinion is, that the death of the patient was due to the entrance of air into the hepatic vein. For even had air entered the portal vein, I cannot see how it could possibly have arrived at the patient's heart, after traversing the minute capillaries of the glandular structure of the liver, which it would be forced to do, in the form of sufficiently large air bubbles as would suffice to prove fatal to life. In the case of the hepatic vein, on the other hand, it would be quite different, as between its trunk and the heart there is no intervening net-work of fine capillaries to subdivide the air globules. However, be that as it may, there was I knew no necessity of my running this risk. All that was necessary in order to avoid it being to take the precaution of guiding the point of the trocar in such a direction as would preclude the possibility of its encountering the large blood vessels which are situated in the neighbourhood of the transverse fissure. Consequently, all I had to do to prevent the entrance of air was to direct the point of the instrument while penetrating the organ in a line between the centre and the upper convex border of the liver, in which position there are exceedingly few, if any, large vessels at all. The danger the patient ran in thus performing the operation I, consequently, considered to be but trifling when compared to the advantages, I was sanguine enough to anticipate would follow upon the free extraction of blood from her inflamed liver. Looking forward, then, with confidence to the result, no sooner had I pushed the instrument home to its hilt, than I began slowly and deliberately, with a rotatory movement, to withdraw it, in the hope that sufficient blood would ooze from the wounded vessels into the channel left by the receding

trocar to reward my efforts. Nor was I doomed in this hope to be disappointed, for scarcely had an inch of the trocar been withdrawn when blood flowed from its orifice abundantly. So all that had now to be done was simply to let it flow. And let it flow I did, until twenty ounces had come away. What was the result? Did the patient suffer much from the operation? or did she die? Neither. Here is what Dr. Walker says in his report of the case :—

"From that day the liver became gradually reduced in size. With the aid of tapping and the administration of the resin of copaiba the ascites, and general anasarca, disappeared, and by the beginning of December the patient was already able to walk out."

I have yet a supplement to add even to this favourable report. The operation was performed on the 4th October, at which time the patient, by Dr. Walker, by me, by her husband, and by herself, was considered to be in a perfectly hopeless state, yet, as Dr. Walker tells us, within two months from that date, she was able to walk out. But, in addition to this, on the 20th of December she walked all the way from Notting Hill to my house, a distance of nearly three miles; and said she "felt perfectly well, only a little weak and stiff from the walk." If the history of this patient, and the result of the operation be not sufficient to encourage the performance of hepatic phlebotomy in suitable cases, I know not what will. So I will offer no comments upon the subject; but confidently leave the case as it stands to the consideration of the advanced school of thinkers among my medical brethren. Feeling certain, that although this is but one case, as everything must have a beginning, and all know that a single positive result is more valuable in deciding a question than a thousand negative ones, they will not fail to give it the attention it merits.

Atrophy of the Liver—Cirrhosis—a Sequel to Hepatitis.

It is commonly said that we live to learn. I think it might be almost said with equal justice that we live to un-learn. This is at least most assuredly true in things medical, and more especially so in matters connected with liver pathology. For no one—as I said in my opening chapter—who has seen much liver practice, and reflected on what he has seen, can possibly have failed to observe that many of the hepatic dogmas he learned from his teachers and his text-books are very far from being consonant with fact. Notwithstanding that several have already been alluded to, it behoves me yet to call attention to others. The first is, that our teachers and our text-books alike err in telling us that atrophy of the liver is a rare affection, except when it occurs in the form of what is known as drunkard's liver—commonly called cirrhosis. My experience has taught me exactly the reverse; for if we omit the disease named acute, or yellow, atrophy of the liver, which is undoubtedly a comparatively speaking, rare affection, atrophy of the gland, both in its chronic and sub-acute forms, is continually being met with. And this will not be considered surprising when I point out that atrophy of the liver, in one or other of these forms, is the invariable sequel to every case of inflammatory enlargement of the organ that has existed sufficiently long to produce induration.

No matter whether the inflammation has been induced by dram-drinking, gluttony, gall-stones, embolisms, chills, malarial or epizootic disease-germs, or anything else. Every liver that has been for a length of time enlarged invariably shrinks, and, if the patient lives long enough,

ends in becoming atrophied. By that term I simply mean smaller than natural. The term cirrhosis I might here employ, but unfortunately it is not explicit enough for my purpose. From the fact that now-a-days, although many speak of the organ as being cirrhosed when it is simply atrophied, there are an almost equal number of others who employ the word to indicate a condition of hepatic induration only, and in this sense speak of hypertrophic, as well as of atrophic, cirrhosis. While, again, the Registrar-General, in his reports, uses the word cirrhosis solely with reference to the condition of liver usually supposed to result from intemperance in alcoholic drinks. As may be inferred from his saying, at p. xv. of the Supplement just issued (1885), "that cirrhosis of the liver is the term which the practitioner employs to denote the consequence of alcoholic excess," having previously stated that "diseases of the liver afford, probably, the best available measure of the extent to which alcoholic excess prevails in the country." With such opposing employments of the term then, I think, for the sake of clinical perspicuity, it would be better for us to cast aside the word cirrhosis—which no one either knows the origin or pathological meaning of—(*a*) and simply use the terms atrophy; and hypertrophic, and atrophic induration, which specifically indicate the respective conditions they are intended to denote, as well as include all the varieties of cirrhosed, hobnailed, nutmeg, contracted, and dram-drinker's liver spoken of by our old authors, without, at the same time, entailing upon us the acceptance of any of their erroneous theories.

(*a*) Neither the Latin word *cirrus*, a curl or lock of hair, nor the Greek word *kirrhos*, signifying tawny, in the slightest degree defines the pathological condition of the liver which at least one of them is supposed to denote.

Such, for example, as they embodied in the term dram-drinker's liver. A more inappropriate name, considering the pathological condition to which it was attached, it being scarcely possible to conceive, seeing that the most typical pathological forms of it, as defined by them, are not only met with in the temperate adult's, but are even to be found in the milk- and water-imbibing babe's liver. For just as hepatitis may occur at any period of life, in like manner, not only may chronic atrophy, but even hardened, lobulated, and hobnailed livers exist in infants. So similar, indeed, are these livers in appearance to the so-called characteristic hobnailed cirrhosed livers of dram-drinkers as to be utterly indistinguishable from them.

An atrophied liver—that is to say one that has been hitherto described under the name of "shrunken" or "cirrhosed"—may indeed be pre-natal; for as atrophy of the liver is, sooner or later, the inevitable sequel of obstruction of the common bile-duct, and an imperforate duct is oftentimes a congenital abnormality, there is frequently met with atrophied livers in new-born children. More than this, it has actually been found that children at the breast—from a week or two old and upwards—who have succumbed to jaundice the result of a congenital deficiency of the common bile or hepatic duct, have had cirrhotic livers in the widest sense of the word. The whole secreting substance of the organ having been found to be interspersed with, and enclosed in dense bands of hypertrophied and hardened fibrous tissue. The nodulated so-called "hobnail" appearance of the surface of the liver in these cases is due to an irregularity occurring in the shrinking of the different tissues of the organ. The fibrous parts, or more correctly, histologically speaking, the intercellular (now hyper-

trophied) connective tissue, shrinking not only more rapidly, but more completely, than the secreting hepatic cells, small lobuli are thereby formed in consequence of the puckering in, as it were, of their surrounding connective tissue, producing the nodulated appearance, not at all unlike a door studded with globular-headed nails.

That the morbid anatomical appearances which I call lobulated, nodulated, or hobnailed atrophy, according to the dimensions of the projections, should come about after a preceding inflammatory enlargement of the organ is easy enough to understand, when it is remembered that during the inflammatory stage the connective tissue becomes greatly hypertrophied, and, by the pressure it exerts on the secreting cells, interferes not only with its own, but at the same time with their proper nourishment, and thereby induces degeneration of tissue. So that, ultimately, not only the normal smooth appearance of the organ may be entirely lost, but it may shrink to even one quarter of its original size. In some cases so small is the liver that no dull sound is said to have been elicitable from it. I have never met with a case where I could not succeed in getting a dull note; but I have certainly met with more than one where the dull, perpendicular area did not exceed more than an inch in extent. As it requires considerable experience to detect an imperfect dull liver-sound, I may as well call attention to the fact that there are several reasons why the hepatic percussion note may be obscured and lead to an erroneous diagnosis, when there is in reality but a moderate atrophy of the organ. These are:—

1. Along with a chronically atrophied liver there is usually ascites, and when fluid is present in the abdomen it so interferes with the hepatic tone as to render it, if feeble, almost imperceptible.

2. When the liver is diminished in size, if there happens to be an accumulation of gas in the stomach, transverse colon, or peritoneal cavity, the tympanitic tone elicited by percussion from the gas may almost completely mask the dull sound produced by the small liver.

3. Even when the liver is of normal dimensions, and there is no great amount of gas in either the stomach or intestines the dull hepatic area may be apparently abnormally small for the following reasons:—

(*a.*) The position of the liver in the abdomen is different in different people—in some being almost completely under the ribs.

(*b.*) When making a diagnosis from data obtained from the size of the liver in women, it must not be forgotten that in consequence of the distortion produced by tight-lacing the conformation of a woman's chest is quite different from that of a man's, and the position of the liver is therefore different. Being often in a woman from one to one and a half inches lower down in the right hypochondriac region, and nearly quite as much below the right nipple than in a man.

(*c.*) The long hepatic ligaments already spoken of at p. 65 occasionally give rise to what are called dislocated, moveable, or floating livers, and some of these sink so far backwards and downwards in the abdomen, when the patient is on his back, as to admit of a loop of tympanitic intestine intervening and completely masking the dull hepatic sound, and lead to the idea of the liver being atrophied when it is not.

(*d.*) It is well also to bear in mind that not only does full stomach materially interfere with percussion,

but that when the patient is in an upright position the liver lies so close to the abdominal parietes that if the examination be made while the patient is standing, even a marked atrophy of the liver may escape detection.

Symptoms of Hepatic Atrophy.

As the disease advances, the patient slowly and gradually loses flesh and becomes weaker and weaker, simply as the result of mal-nutrition, from the partial arrest of the liver's functions. At the same time the complexion assumes a dusky, or sallow hue. Not a jaundiced appearance, unless the case be complicated with gall-stones or some other jaundice-exciting cause, which it often is.

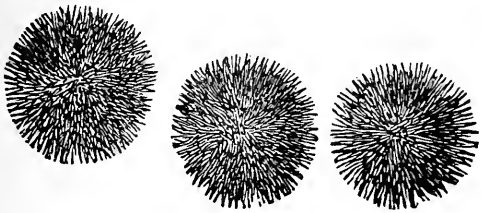
The digestion becomes impaired, the bowels irregular, and sometimes we are presented with the strange anomaly of grey, putty-like, almost pipeclay coloured stools, without either jaundice or bilious urine; a condition of things which long puzzled me, as it had done others, to account for. At length I imagined I had obtained a clue to the mystery why the stools were without bile, and yet neither jaundice nor bilious urine was present, from my noticing that in nearly every one of the cases in which the non-bile looking stools occurred without there being any appearance of jaundice, there was almost invariably after a succession of grey or putty-like stools—one, two, or sometimes three—with distinct evidence of bile in them; and this reappearance of bile was again immediately followed by a succession of the grey stools. Reflection on this fact led me to the opinion that this strange phenomenon must be due to the atrophied liver secreting a small quantity of bile,

which was, as in health, stored up in the gall-bladder, but from the amount of bile daily formed being insignificant, it took some time before the gall-bladder got sufficiently filled to have its peristaltic action excited by its contents, and thereby made, as in health, to expel them. Hence no bile would pass from it into the intestines during the intervals of the filling process. Consequently there would be periods when no bile appeared in the stools, as well as periods when it would. This appears to me to be the probable explanation of the anomaly; and it is further strengthened by the fact that the patients are usually exceedingly flatulent—the gas coming, no doubt, from the food fermenting in the digestive canal in consequence of the absence of its natural anti-septic bile. As beyond these not very striking signs there is little or no disturbance to the general health, the atrophying process in the liver sometimes advances so insiduously as to remain unsuspected, by either doctor or patient, until some change takes place in the symptoms and calls special attention to his state. Such, for example, as a sudden outburst of hæmorrhage, either from nose, mouth, anus, urethra, or vagina, or the gradual supervention of ascites and dropsy. As these are formidable complications in liver disease, instead of saying anything more here regarding their connection with hepatic atrophy, I shall, after finishing this chapter devote two special ones to their consideration, to which I beg to refer the reader, pp. 90 and 105.

The condition of the urine furnishes us with important information in cases of chronic, as well as acute, atrophy of the liver. For example, as I pointed out in my book on Jaundice (1863), in mostly all severe cases of atrophy of the liver both tyrosin and leucine are met with in the urine. All that is necessary to find them

being to evaporate the urine slowly in a water bath until it becomes concentrated. The tyrosin appears in the form of round spiculated balls as represented in Fig. 2.

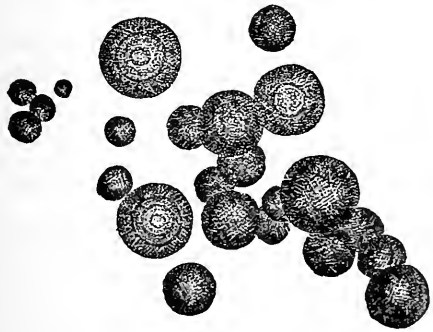
Fig. 2.



Spiculated Balls of Tyrosin.

When these are redissolved and recrystallised, they assume the form of needles and stellate crystals. Leucine, on the other hand, while it, too, appears in the form of balls, never assumes a spiculated, hedgehog-like appearance, but as dark globules like carbonate of lime, from which they are easily distinguished by their not effervescing on being treated with nitric acid.

Fig. 3.



Dark Globules of Leucine.

Treatment of Chronic Atrophy of the Liver.

In the treatment of early diagnosed cases of atrophying liver it is the cause which ought to be attacked: but where the organ has become decidedly atrophied before the patient comes under treatment, no matter whether the contracted state of the liver be the result of dram-drinking, malarial hepatitis, gallstones, or other cause, regulated diet and regimen are the chief means to be adopted to retard the progress of the disease. To aid in that object the following suggestions may be found useful:—

1st. Give a due proportion of animal and vegetable food. The former *always fresh*, the latter thoroughly cooked. A good combination is milk and eggs along with tapioca, corn flour, ground rice, sago, or arrowroot, in the shape of puddings.

2nd. Moderate exercise in the open air; living in a dry situation; sleeping at a high altitude, and, when the weather permits of it, with the bedroom window open.

3rd. The careful avoidance of cold baths, or any other sudden change of temperature which will give a chill or shock to the system.

4th. Night and morning brisk cutaneous frictions with flesh brushes.

5th. As regards medicines: Tonics and vegetable pure gatives are to be used according to circumstances. Mineral acids I have ceased to employ from never having seen any benefit follow their administration, while their constipating effects always require to be counteracted by other medicines, otherwise the hepatic congestion is favoured instead of diminished.

Lastly. Both the flatulence and the mal-digestion of the food may in a measure be counteracted by artificially supplying the place of the deficient bile by daily administering two or three of the five-grain gelatin-coated pig's bile pills manufactured by Parke, Davis, & Co. It is essential that the bile pills be good; otherwise they will produce no beneficial effect. Their proper action is indicated by improved digestion, diminished flatulence, and the appearance of bile in the stools. As adjuncts of digestion, enzymes (more especially that of pancreatine, not pancreatic emulsion), are likewise of great service. For the stomachal, as well as duodenal, digestion is oftentimes so greatly impaired that the food is voided by stool, but little altered in appearance from what it was when eaten.

From cases of hepatic atrophy being chronic, and I am frequently asked, both by doctors and the patient's friends, how long it is likely for the sufferer to survive, it may not be altogether unacceptable to some of my *confrères* if I say a word or two on the subject of prognosis.

Life, of course, is proverbially uncertain, being rendered all the more so, not only by the constitutions, but the conditions of patients being different. There are, however, one or two things which may lead to our forming a tolerable correct estimate of the probable duration of any given case. The first is the rapidity with which the atrophying process is advancing. The second, the size of the liver at the time the prognosis is demanded. When the atrophying process is rapid the patient's life is to be calculated by weeks. If it be slow, by months. On the other hand, if the atrophying process has been arrested, even should the hepatic dulness be within a couple of inches, if there be no dangerous com-

plication, the patient, with careful attention, may be kept alive for years.

The Ascites and Dropsy of Liver Disease.

When ascites supervenes, as it frequently does, in the course, either of Inflammatory Hypertrophic Induration or its sequel—Atrophic induration of the liver, it is invariably of bad omen. Curable it sometimes is; but not often. Much more curable in hepatic enlargement than in hepatic atrophy. In the latter case, indeed, only when the atrophying process can be not alone arrested; but the liver tissues induced to regain somewhat of their normal state. In order to be able to accomplish this, both continuous and judicious treatment is necessary. Sometimes even, however, when to improve the condition of the liver, is beyond the power of medicine, we are yet able to impede the fatal march of the disease, and keep the patient not only alive, but in comparative comfort for many months, occasionally, though rarely, even for years.

As in order to treat rationally, in contra-distinction to empirically, it is necessary that the nature of the disease be thoroughly understood, I shall preface my remarks on treatment with a few words on the pathology of hepatic ascites.

Firstly: In both enlarged and contracted livers the accumulation of fluid in the abdomen is due to precisely the same cause, namely, the hepatic tissues constricting the vena cava. Consequently, ascites never occurs except when the portion of the liver tissues actually surrounding the vein is diseased.

Secondly: The fluid filling the peritoneal cavity is

effused from the coats of the vena cava, and other abdominal veins, and is nothing else than blood serum. Consequently it is highly albuminous, usually depositing after boiling, and the addition of nitric acid, about half its bulk of coagulated albumen. Its specific gravity is in general 1011-1012.

Thirdly: It is a clear, opal-looking liquid, except when jaundice, hæmorrhage, or peritonitis is present. In the first case it may be of an amber colour, or even of a sufficiently orange tint to stain objects yellow; while in the second of a pink or even blood-like hue. When there is peritonitis present, the fluid is turbid, milky-looking, and sometimes contains flocculi.

Fourthly: Neither cholestearine, urea, nor uric acid crystals are found in it. A small quantity of chloride of sodium is, however, in general, present.

Fifthly: The rapidity of its secretion varies exceedingly in different cases. In some it is so slow that the increase, from day to day, in the size of the abdomen is almost imperceptible, especially at the beginning. In others it proceeds with astonishing rapidity. More especially after it has continued for some time.

The ordinary amount of serum exuded in the average run of cases I estimate to be from 16 to 36 ounces per diem. But in bad cases the quantity may be half a gallon and upwards. In the case of a gentleman, æt. 38, who I saw in consultation with Dr. Bannister, over a gallon of pure ascitic fluid was secreted in a single day, and no less than 12 gallons were withdrawn from his abdomen in 13 days. The total quantity again removed from a patient during a long illness is really remarkable. For example, a lady, æt. 37, who I saw to-day (13th January) has been tapped 56 times since October, 1884 when, on that occasion, 17 quarts—that is to say, 4¼

gallons—were taken away. Since then she has been, on an average, tapped every 10 days. Dr. Dumbleton, who first brought the case to me in August last, has furnished me with the following particulars:—From the first till the twenty-third tapping from 14 to 15 quarts were each time removed. On the next thirty occasions the quantity averaged 12 quarts; while on the last three it has been $7\frac{1}{2}$ quarts in 12 days, and the total quantity withdrawn in the 56 tappings, during the whole fifteen months she has been ill, has reached 160 gallons. That is to say three hogsheads, equal in weight to half a ton, of albuminous serum have been artificially removed from an average sized ($9\frac{1}{2}$ stone) woman's abdomen; and that, too, not only with impunity, but with actual advantage; for the amount daily secreted is gradually becoming less and less.

The case is one of atrophy after hepatitis, consequent upon several years' residence in India. When I first examined the lady the hepatic dulness in the right perpendicular nipple line was $1\frac{3}{4}$ inches. Now it is $2\frac{1}{4}$, and gradually as the liver is regaining its proper size the secreted ascitic fluid is proportionately becoming less; for while at first it is calculated to have formed at the rate of 3 pints per diem, now it does not exceed 25 ounces. At the same time, the patient has gained over a stone in weight, and not only looks, but says she feels, quite a different being. The most interesting clinical feature in the case has yet to be told. Namely, that the day after the last withdrawal of 15 quarts she was up and playing on the piano to amuse herself; and that is not all, Dr. Dumbleton drew off $7\frac{1}{2}$ quarts of fluid last night at 9.20, and at 11.20 this morning—that is to say, exactly 14 hours afterwards she walked into my room, having driven from her house at Bayswater in a cab. Here we

have a striking instance of the toleration of the operation of tapping, as well as of the advantage of getting the patient out of bed as soon as possible after the operation. According to my experience, the lying in bed, when there is no necessity for it, only weakens the patient. So I always recommend them to get up after the tapping as soon as they feel sufficiently strong to sit in a chair. I may mention that, in the majority of instances, hepatic ascites is easily diagnosed from renal and cardiac dropsies by the following signs :—

a. In hepatic ascites alone are the superficial abdominal veins dilated.

b. Only in renal and cardiac dropsy does the œdema extend above the waist, except in hepatic cases, when, for a time, the patient has occupied the recumbent position, the dorsum becomes œdematous, and effusion takes place into the pleura.

c. Although the urine may be albuminous it is never in an uncomplicated case of hepatic dropsy of a lower specific gravity than 1012.

d. In hepatic ascites, even without jaundice, the stools do not always contain bile. Sometimes they are grey, sometimes of a pipeclay colour, and accompanied with much flatulence.

The greatest danger in cases of ascites arises from sudden stoppage of the heart's action. In consequence of the pent-up abdominal fluid pushing the diaphragm against the heart's apex, and arresting its movements. There is even great danger when the abdomen is not quite full of fluid, if, as often happens in hepatic cases, for the reason assigned at p. 86, the stomach gets filled with wind ; for unless the patient can speedily get rid of the wind by belching, it presses up the diaphragm and

stops the heart's action as effectually as the ascitic fluid itself does.

For this reason, I always order a stimulant anti-flatulent medicine, of some kind or another, to be kept in readiness, so as to be given to the patient the moment a feeling of syncope is experienced. Of these remedies one has a large choice. Many of the etherial oils are useful. Particularly so the oils of anethi, cajeputi, carui, juniperi, menthæ, and pure terebene. All of which may be taken in the form of drops on a piece of sugar, or made into a mixture along with carbonate of ammonia and spirits of chloroform, or tincture of cardamoms. Creasote and ginger, sulphite and sulpho-carbolate of soda, as well as assafœtida and camphor, are likewise useful. What will suit one patient will be found not to suit another, and what is still more annoying is, that all of them soon lose their beneficial effects, and consequently require perpetual changing.

In consequence of the impediments caused by the ascitic fluid both to free cardiac and pulmonary action and the sudden dangerous results not unlikely to arise from them, it is true prudence to tell the patient's attendants to give timely warning of even the occasional appearance of palpitation, difficulty of breathing, feelings of syncope, or sickness; for as soon as these begin to show themselves, surgical treatment becomes necessary.

Treatment of Ascites.

Ascites is, in the first instance, to be counteracted by the administration of diuretics. The most useful I have found to be copaiba, squills, iodide of potassium, nitrate of potash, sweet spirits of nitre, and an infusion of broom

tops. A selected mixture of these should be given every three or four hours in weak, warm linseed tea, flavoured with lemon. I have further found great benefit to follow upon the use of a pill composed of copaiba resin, quinine, and extract of aloes, given equally frequently, and before food. As a striking illustration of the truth of this remark, I will briefly relate a case which is at this moment under treatment, as the results I have to record, in as far as they go—even imperfect though they be—cannot fail to impress the value of this method of treatment on the mind of the reader.

The patient is a strongly-built man—at one time a celebrated athlete—of 48 years of age, who says he never was ill until five months ago, with one exception, and that was in 1854, when he was roughing it at the diggings in Australia, where he had an attack of what they called "digging-fever." For two months before consulting me he had been under the care of a practitioner in the Commercial Road, who he left on the 30th December last, on account, he said, of his getting worse instead of better under his care, and his doctor not proposing a consultation. When he came to me his case did not appear a very promising one for treatment, as, not only had he an enlarged liver, with ascites, but likewise a diseased heart. His pulse was 102, and his respirations 34 per minute. His lower limbs were œdematous, and his abdomen so full of ascitic fluid as to measure at the navel 70½ inches in circumference. His liver reached as high up as the right nipple, and extended 11½ inches downwards. I prescribed for him a pill consisting of quinine, croton-chloral, extract of aloes, and copaiba resin, and a plaster to be applied over the heart. On the 16th January—that is to say, exactly after seventeen days' treatment—a marvellous change for the better had

taken place. Not only were pulse and respirations greatly improved, but the girth of the abdomen was now only 56 inches, and he said he was "feeling quite a different man." A decided change had also taken place in the liver; it measured 9 inches in the nipple line. But what surprised me most was to hear that he was passing in the twenty-four hours ten pints of urine, while only drinking five pints of liquid! In all my wide experience of hepatic and urinary diseases, I never before met with a patient who, like this one, passed 200 ounces of urine in twenty-four hours while only drinking 100 ounces of liquid. The urine he passed in my room was of a dark straw colour, and of a specific gravity of 1015. After boiling, and nitric acid being added, it deposited half albumen. The condition of the heart was unchanged. The pulse, however, was now only 96, and the respirations 25 per minute. 24th January—that is to say, after twenty-five days' treatment—the improvement still continues. He can now get his boots on, and his girth is only 54 inches—that is, 16½ inches less than it was twenty-five days ago. His breathing, too, is much better. He can now mount twelve steps at a time without having to stop to take breath, while before he could only go up three. The respirations are still, however, 23 per minute; his pulse is 88. He is not making so much water, only about 140 ounces; neither is he drinking so much liquid—not more, he thinks, than about from 60 to 70 ounces. So, it may be said, that he is still passing double the quantity of liquid he is taking. His bowels are opened twice or thrice every day.

This is an exceptionally favourable case for the character of diuretics. Unfortunately there are but few like it, and notwithstanding the improvement which has so speedily taken place, both in the condition of the liver

and the ascites, I am naturally doubtful if it will continue. In such cases, as a rule, tapping has to be had recourse to, and when once it is begun it has to be gone on with, for which reason I never advise a patient to be operated upon for the first time until the presence of the fluid causes inconvenience by pressing the diaphragm against heart and lungs. And when the tapping is done, I always recommend the fluid to be drawn off slowly and completely by a three or four-inch long Potain's syphon trocar, or a guarded-pointed aspirating needle of the diameter of a No. 2 or 3 sized English catheter, through which I find the fluid usually flows at the rate of a pint in six or seven and a-half minutes. A syphon tube attached to the instrument ought to be used. It is preferable to an aspirator, as the act of aspiration often draws the intestines against the orifice of the canula, and arrests the flow of the fluid.

Notwithstanding the facility with which the operation of tapping is performed, as well as the rarity of any disagreeable consequences following it, I think it is always well to refrain from speaking to the patient's friends of the operation as being a trifling one, for a fatal peritonitis may be the sequel, as the following case shows:—In 1880, Dr. Nixon showed at the Dublin Pathological Society specimens from a man, æt. 45, who died after tapping for ascites consequent on cirrhosis of the liver. In a few days—after several quarts of fluid had been withdrawn—a diffuse erysipelatous blush appeared round the seat of puncture, general peritonitis supervened, and the patient died. At the necropsy an abscess was found between the peritoneum and abdominal wall.

There is another danger which I must allude to, as it is not, as far as I am aware, mentioned in books. That is, the accidental opening of a large vein while inserting

the trocar, and thereby giving rise to considerable hæmorrhage. On one occasion I was told that the bleeding was so severe as to necessitate the surgeon's passing a needle and thread beneath the vein, and securing it by ligature. The abdominal veins in all cases of hepatic ascites, as I before said, are greatly dilated. Care ought, therefore, to be always taken to look well that the trocar be not thrust through one.

I need hardly, I think, say that in tapping fat people a trocar not less than three inches in length is absolutely necessary, as it is no uncommon thing for the abdominal parietes to be two and a-half inches thick in them; hence the employment of a short trocar will only lead to disappointment. Even after the operation has been supposed to have been most satisfactorily performed, I have more than once seen great annoyance and discomfort caused to the patient from the puncture-wound not closing; and as a natural consequence an oozing of ascitic fluid taking place from it. No disadvantage arises from this discharge, but, on the contrary, an advantage, except from the inconvenience the dampness occasions, which can usually be provided against by keeping a sponge or a thick pledget of absorbent cotton over the orifice of the puncture, but the cause itself had much better be avoided. There are one or two hints, therefore, which, I think, I may venture, even as a physician, to give young surgeons as regards the modes of procedure in tapping. They are:—Firstly, avoid the now common practice of making a preliminary incision in the skin before inserting the trocar, for if a sharp-pointed, small, No. 2 or 3 sized trocar be used, as it ought to be, the incising the skin, so far from diminishing the pain, only makes two separate pains instead of one, while it doubles the time required in making the puncture.

Secondly, it slightly increases the size of the cutaneous wound.

Thirdly, it possesses not one single advantage, present or prospective, to either surgeon or patient.

Fourthly, puncture through the recti muscles in preference to the linea alba, as there is then not only far less chance of leakage, but the wound heals quicker. This is not quite such an unimportant remark as some may imagine. A sad case came under my notice a week ago, which forcibly showed me the importance of attending to the above seemingly trifling details. On the 4th December I was called to a gentleman, æt. 42, living not very far from my own house, whom I ordered to be at once tapped. I heard and saw nothing more of him until the evening of the 6th of January, when I was suddenly summoned to his bed-side, the messenger saying "he had burst." When I reached the house I met his medical attendant, who told me he had tapped the patient on three occasions, drawing off respectively seven, ten, and seven quarts. On each occasion he had operated in the linea alba with an ordinary ascitic trocar the size of a No. 10 English catheter. Eight days after the last tapping the seat of the puncture suddenly gave way, and ever since then fluid had been running from the wound at the rate of half a gallon a day, saturating the bed-clothes, and keeping the patient in great discomfort. I advised the insertion of a drainage-tube in order to keep the bed dry, but unfortunately, before it could be of any service, the patient died.

The occurrence of accidents like this occasionally leads to the operator being suspected of bungling, so I will allude to one or two other points, which, I think, though apparently equally trifling, it is wise for the surgeon to attend to. In closing the wound, use neither large pieces

of sticking-plaster nor collodion lint pledgets, as both are apt to excite considerable irritation. I have seen an erythema follow on the employment of the one, and a loss of cuticle succeed that of the other. The following piece of advice as regards the mode of closing the wound is, therefore, always the one I give, namely:—If the puncture has been made (as I have advised) through one of the recti muscles, all that is requisite to ensure its proper closure is to cover it with an inch and a-half, square-sized piece of sticking-plaster, over which place a pledget of lint, and bind up the abdomen with a broad-tailed bandage. The object of the lint is two-fold—firstly, to keep the sticking-plaster safe in its position; and secondly, to act as a compress and support to the now lax and flabby abdominal parietes.

I have yet to call attention to another not generally known fact which might, if not understood, cause considerable embarrassment to an operator, and that is, finding no fluid follow the entrance of his trocar, notwithstanding the abdomen being full of serum. I was once present when this occurred. The patient was a lady, æt. 36, labouring under ascites from an enlarged liver, consequent on an attack of hepatitis, induced by impacted gall-stone. She had been tapped a week previously by Dr. Diver, and two gallons of serum drawn off. Having already tapped her in the linea alba, he proceeded to perform the operation exactly in the same way on the right side, at a point midway between the umbilicus and spinous process of the crest of the ilium; but on withdrawing the stile from the canula, to his astonishment not a single drop of serum came away—only a few drops of blood. The canula was withdrawn, and the trocar reinserted about a couple of inches nearer the mesial line, but again with a negative result. He then introduced the trocar at a

level of about an inch and a-half above the umbilicus on the right side, when serum flowed. A couple of months afterwards the cause of the mishap was revealed at the post-mortem. The patient, who had, several years before, suffered from inflammation of the uterus and its appendages, had apparently had a recent attack of peritonitis, for the intestines in the vicinity of the unsuccessful punctures were found to be agglutinated together into a compact mass. There was therefore no doubt that the point of the canula had lodged itself among them instead of in the free abdominal cavity; hence the reason why no serum was obtained from the peritoneum in that situation. I have a still more curious *contretemps* of a somewhat similar nature to relate, although its cause was entirely different. Within the last fortnight I saw a lady, æt. 45, along with Mr. E. C. Barnes, of Hammersmith. She was suffering from ascites, consequent upon a hypertrophied indurated liver, and as there were symptoms indicative of impending hæmorrhage, it was deemed advisable to tap her. Mr. Barnes, who has had considerable experience in such cases, proceeded to tap her a little below and somewhat to the left of the umbilicus. A pint of serum flowed freely away, when all of a sudden the flow ceased. Thinking that the canula had for some reason or another got stopped up, he withdrew it and inserted another of twice its diameter; but instead of serum now flowing, blood alone came away. He withdrew the instrument, closed the wound, and examined the abdomen, and at once discovered a hard tumour exactly behind the seat of puncture. I saw the patient on the following day, when the tumour was most palpable to the touch, although before the pint of serum was drawn off it was undiscernible. The opinion I therefore formed was that the hard mass

was a tumour of the left ovary, which had come forward against the abdominal parietes so soon as the intervening layer of liquid was withdrawn. It was therefore arranged that the patient should be tapped on the other side of the abdomen and higher up. This was on a Saturday, and the operation was to be done on the Monday, but on the Monday forenoon the patient was suddenly seized with hæmatemesis, and expired before Mr. Barnes reached the house. A post-mortem was made, and besides the enlarged indurated liver was found a solid utero-ovarian tumour, immediately behind the seat of tapping. The abdomen contained a gallon of ascitic fluid. Thus we see how, notwithstanding that a patient's abdomen may be full of serum, a series of extraneous circumstances may prevent its withdrawal by a trocar introduced at certain situations, although the peritoneal cavity may be readily and completely emptied of its ascitic fluid should the tapping be done at another part of the abdomen. Twice I have encountered cases where the ascitic fluid in the abdomen has seemed to have undergone a process of consolidation. The one in the woman, æt. 36, the other in a man of 48 years of age. In both patients, when they came to be tapped a few days after a distinct liquid wave had been obtainable all over the abdomen, not only was the liquid wave no longer perceptible below the umbilicus, but trocars introduced there failed to bring away any ascitic fluid whatever, although when the trocar was introduced above and to the side of the umbilicus, fluid flowed freely. This condition of things seems only comprehensible on the supposition that, in sub-acute peritonitis, some kind or another of consolidation of the ascitic fluid takes place. Otherwise to me the matter is at present unaccountable. I would gladly have information on the subject from

anyone who has ever encountered one of these peculiar, as well as exceedingly rare, cases.

As I am trying to give all the information I have obtained from an extended experience of hepatic ascitic cases, it may be as well for me, perhaps, here to allude to a mistake which many surgeons commit in tapping weak patients—that is, making them sit in a chair during the operation, under the erroneous idea that the fluid is more easily withdrawn from the abdomen when the invalid is in a sitting than in a reclining position. The sitting position during the operation is highly objectionable for the following reasons:—

Firstly, because the fluid flows equally freely away when the patient is in a recumbent attitude.

Secondly, from the important fact that fainting is much more likely to be induced in a sitting than in a lying condition.

Thirdly, the fatigue of getting weak patients out and back again to bed ought always to be avoided.

Fourthly, I never knew a single patient who had gone through the experience of having been both tapped in bed and in a chair elect of his own accord the latter position for a second time ; but, on the contrary, object to it.

There is a further little piece of advice which I would venture to give, even at the risk of its being probably considered a trivial one by some of my readers little experienced in the tapping of patients. It is, that, as the operation seldom lasts less than an hour, and very often three hours, it is good policy, more especially when timid patients are being operated on for the first time, not to leave them too much to their own thoughts during the operation, or the result may be the supervention of a fainting fit. It is well, therefore, for the attendants,

instead of sitting like mutes in gloomy silence, to engage now and then, amongst themselves, in a little entertaining conversation, as it is sure to have a salutary effect on the invalid, and not unlikely prevent him or her fainting from pure emotion; which has, I believe, happened more than once, though it has been attributed to the effects of the withdrawal of the fluid, instead of to its real cause.

After having cited the fatal case of peritonitis, following upon tapping, it will be scarcely necessary for me, I think, to give the advice that one ought always to be scrupulously particular to operate with perfectly clean instruments, as well as to carefully lubricate them with antiseptic carbolised oil. No anæsthetic is required in an ordinary case of tapping for ascites. Although it is usually only the first tapping which causes much discomfort to the patient, and it is always wise to delay it as long as possible, from a pretty extensive experience of these cases, I have come to the conclusion that the operation ought to be had recourse to so soon as the patient complains of shortness of breath, feelings of sickness, or of syncope, lest a sudden and unexpected failure of the heart's action should occur. And when once the tapping has been had recourse to, and the patient has learned not to dread the operation, there is no longer any necessity for delaying its repetition for a single moment after the first symptoms of cardiac or respiratory discomfort manifest themselves.

Moreover, always take the precaution to have the urinary bladder emptied before operating, in order to avoid all risk of puncturing it during the operation.

Finally, after having gone so fully into the subject of tapping, I think I cannot do better than conclude by

saying that after the operation has been performed the patient ought to have some refreshing, slightly stimulating beverage, and immediately afterwards placed in a position to fall asleep as soon as possible.

Hæmorrhages Connected with Liver Disease; Bleedings from Nose, Mouth, Urethra, Vagina, and Anus; together with Cerebral and Hepatic Apoplexy.

This is an almost untrodden field of inquiry; for although from time immemorial hæmorrhage from the bowels in cases of atrophy (cirrhosis) of the liver have received attention, and many cases of hæmorrhage from the nose, mouth, urethra, vagina, and anus have been recorded and commented upon by various writers as having been met with in a variety of other forms of liver disease, yet no one has attempted to classify, generalise upon, and deduce inferences from the frequent occurrence of profuse, and sometimes fatal bleedings, as a whole, in the course of hepatic affections. This has most probably arisen from the fact that many of them do not always appear in the course of apparently similar cases; so that the occurrence of several of them has been regarded more in the light of an accidental coincidence than as they are in reality, as I shall presently show, the direct result of cause and effect.

The pathology of some hæmorrhages from hepatic causes is easy of comprehension, that of others difficult to define. I shall commence with the easiest.

All biliary concretions while attempting to leave the

gall-bladder, or bile ducts, by ulcerative perforation are apt to produce hæmorrhage, on account of their opening a blood-vessel in the course of their transit. Hydatids, by implicating blood-vessels, equally, though less frequently, do the same thing. The blood is usually in such cases poured into the digestive canal, and ejected from the mouth as a hæmatemesis, or passed by stool as a melæna. If there be much blood, and it be rapidly expelled, it appears both in vomit and stool as pure blood. I have seen as much as a quart of perfectly pure blood vomited within a few minutes. The case was that of a lady, æt. 53, who, for the second time, suffered from perforating gall-stone. She also passed a quantity of blood by stool, and yet recovered. The patient was sent to me from Eastbourne by Dr. Habgood. When the blood has been retained for some time in the digestive canal it becomes much changed in appearance from being acted upon by the acid secretions. It then looks like coffee grounds; or, if it remains longer still, it is ejected upwards as "black vomit" and passed downwards as "tarry-stools." I know of one case where a hydatid opened a vessel in connection with the common bile duct, along which the blood flowed into the intestines, and while a portion of it was vomited as pure blood a quantity was passed by anus as tarry stools. The case, unfortunately, ended fatally. At the necropsy a large quantity of blood coagulum was found yet remaining in both stomach and duodenum.

The next set of cases of hæmorrhage in connection with liver disease are those in which there are bleedings from the nose, mouth, urethra, vagina, or anus, in the course of enlarged liver (from malaria, gall-stones, or other exciting cause of hepatitis), atrophy, and cancer. Their pathology is entirely different from the preceding,

and may, I think, be defined, as in the case of hepatic ascites, as the result of the constriction of the vena cava, caused by the disease of the liver in its vicinity.

As in ascites, hæmorrhage, either from hepatic hypertrophic induration or atrophic induration, never occurs except when the portion of the liver around the vena cava is diseased.

It is very easy to understand why hæmorrhages should take place below the seat of constriction, and consequent impediment to the upward flow of the venous blood through the vena cava. For example, from piles, from uterus, and from bladder; but it is not so easy, at first sight, to see why a constriction of the vena cava should, produce, as it so often does, an epistaxis, or an apoplexy.

A little reflection, however, soon solves the difficulty, for in many persons having a predisposition to nose-bleeding, either from possessing a hæmorrhagic tendency or an atheromatous condition of the coats of the blood-vessel, or other cause, the increased tension induced in the arterial system, consequent upon the impediment to the free circulation of the great mass of venous blood suffices to rupture a vessel and let a stream of blood pour forth. A precisely similar pathological explanation may be given for the frequency of attacks of apoplexy in persons labouring under liver disease, either in the form of hypertrophic or atrophic induration. Aye, and that, too, in persons of a spare habit of body, and almost ex-sanguine appearance.

In case some reader fails to grasp the validity of this view of the matter, I will give a most striking illustrative example which fell under my notice some years ago, and did much to clear up my ideas on the subject. The history of the case is briefly this: One day, as I was finishing lunch, the late Dr. Wildbore hurriedly drove up

and asked me to go with him to see a lady who was in a state of syncope from which it was impossible to rouse her. As we drove along he gave me the following history of the case. She was married, 49 years of age, of spare habit of body, and had for some years had an enlarged liver, which he thought might be cancerous. She never had jaundice, but was frequently very bilious looking. She lived well, rather freely, and just as she was rising from the table (about half an hour before he came for me) after partaking of a hearty meal she had fallen down, as he said, in a dead faint. This account of the attack, as she had received no sudden mental shock, suggested to my mind the possibility of one of two things: (*a*) It might be that an over-distended stomach, by pressing the diaphragm against the heart's apex, had stopped its action; (*b*) It might be a case of sudden collapse from internal hæmorrhage, from the rupture of an aneurism. The idea of apoplexy did not enter my mind, from his having said she was only 49, and of spare habit of body. Arrived at the house, which, being near at hand, we did in a few minutes, I found the patient extended on the dining-room sofa, breathing heavily, with a cold and bloodless face. Her pupils were dilated, one much more than the other. The pulse was slow and laboured. I advised the case to be treated as one of cerebral apoplexy, consequent upon the liver disease. The remainder is soon told. Within a short time stertorous breathing set in, and in a few hours the patient died.

I shall now briefly cite another case of apoplexy equally from liver disease, but in its history as different from the preceding as night from day. It occurred in a well-known scientific man, æt. 52. He was of large frame, short necked, and plethoric habit of body. The

very subject in which one would expect apoplexy. Although not a drunkard, he was most assuredly intemperate; as I well know from my having been intimate with him for many years. He came to me on account of his liver, which, when I examined it, was twice its natural size, and as hard as a board. I only saw him once professionally, as, not liking the stringent injunctions I gave him as regards eating and drinking, he never consulted me again. In fact, he had left Sir William Gull for the same reason. Within a year after consulting me he died, and his death was stated in the papers to be from apoplexy. Whether or no there was atheromatous degeneration of the blood-vessels in this case I do not know, but I think it probable that there was, from what I know of a brother of his, who consulted me for profuse and repeated attacks of epistaxis. Mr., now Sir, Prescott Hewett saw him along with me, and such was our united diagnosis.

There is even such a thing as apoplexy of the liver itself. In most of our old text-books, and in a few of our modern ones, liver apoplexy is described as consisting of extravasated masses of blood in the hepatic parenchyma, "the result of congestion generally induced by the morbid changes in the blood, met with in cases of scurvy, purpura, ichorrhœmia, and especially in the malarious fevers of hot climates" (Tanner's "Practice of Medicine," 1861). The condition is undoubted, but the pathology, as here given, is, I believe, quite erroneous. The best example I ever saw of liver apoplexy was in the post-mortem theatre at Vienna. The case was that of a woman in whose liver were a number of dark red coloured, cherry- and chestnut-sized clots of blood, scattered throughout both lobes. The blood had apparently escaped from minute lesions in the twigs of the hepatic

or portal veins, on account of obstructive congestion. The extravasations were probably favoured by a hæmorrhagic tendency; but certainly there was no evidence of any scorbutic or ichorrhœmic taint. This kind of liver extravasations arise, in my opinion, in the same way as the hæmorrhages into the digestive canal, already alluded to.

Hæmorrhages from venous obstruction, the result of a hypertrophic or atrophic condition of the liver, are in no case necessarily visible externally, as the following two examples show.

A girl, æt. 13, too ill to give any history of herself, died in University College Hospital within twenty-four hours of her admission. At the autopsy the liver was not only found atrophied (it weighed only 26 ounces), but so nodulated, lobulated, and convoluted as to look more like a brain than anything else. Her stomach and duodenum contained such a quantity of clotted blood that Dr. Hillier, who made the necropsy along with me, entered the case, with my concurrence, in the hospital-book as one of "cirrhosis, accompanied with profuse hæmorrhage into the stomach and intestines." A remarkable case of hæmorrhage from cirrhosis, into the peritoneum, was brought by Dr. T. A. Wood before the Liverpool Medical Institution in 1884. The short reprint I saw of it stated that—"Four or five years ago, the liver extended two inches below the margin of the ribs. Fifteen months ago, it attained its largest size, reaching four inches below the ribs; since then it gradually receded, until it could not be felt at all. The reason he brought the case forward was on account of the unusual cause of death, which was *hæmorrhage into the peritoneal cavity*, where, at the *post-mortem* examination, a large quantity of clotted blood was found."

It may be well for me here to observe that the nose

bleedings so common in cases of gall-stones, are not due to the mere presence of the biliary concretions; but to the indirect effects they produce on the liver. Namely, the hepatitis they engender, and subsequent shrinking of the organ. I have met with, I should think, no less than a dozen instances where profuse epistaxis has occurred at some time in the course of gall-stone affections. In one case, æt. 62, in particular, which I have had under my care (where 53 gall-stones have been discovered in the stools) during the last four years there have been repeated attacks of nasal hæmorrhage. And on one occasion so profuse and little amenable to plugging was it that the lady's life for some hours literally hung in the balance.

It may be prudent for me further to remark that care must be taken not to fall into the error of supposing that all cases of hæmorrhage from nose, mouth, bowels, or bladder occurring in the course of liver disease are necessarily dependent upon it.

Yesterday, for example, Dr. Walker, of Ladbrook Road, brought to me a little blond girl of three years of age with a large hydatid of the liver, who has occasionally suffered from hæmaturia. But, as he justly remarked, the bloody urine in this case has an entirely different origin than the liver mischief; being in fact the result of renal gravel.

The tarry dejections and black or blood vomit in cases of contagious jaundice (yellow fever) and acute atrophy (yellow) of the liver, are of course the direct result of hepatic disease, in the same way as is the paroxysmal hæmaturia spoken of at p. 67.

Hæmorrhages in the course of liver disease associated with ascites are by no means uncommon. In fact, so frequent are they that if there be the slightest suspicion

of it I always call attention to the probability of hæmorrhage supervening, either from nose, bowels, or stomach in all cases of hepatic ascites. The advantage of doing so was well illustrated in the case I saw along with Mr. C. E. Barnes (p. 101), for within 24 hours after I had mentioned the probability of hæmorrhage taking place into the digestive canal (the lady æt. 45, was suffering from an enlarged and indurated liver), blood began to pass by stool, and a few days afterwards the patient died, as before said, from an attack of hæmatemesis.

I have next to refer to another point. It is one which I have found to be of considerable importance. Namely, the association of jaundice with hæmorrhage. Hitherto I have been treating of cases in which jaundice is more frequently absent than present. Now I have to call attention to the fact that jaundice is sometimes the result of hæmorrhage. For long the association with jaundice and bleeding was thought to be a mere coincidence. Now, however, we are gradually becoming conscious that the two may be co-ordinated together in the direct relationship of cause and effect. The two best cases I know in support of this idea were recorded by Mr. W. Smith, of Clifton.

The first being that of a collier, æt. 24, who, in killing a duck, thrust the knife into his radial artery, from which a profuse and dangerous bleeding ensued. The wound rapidly healed, but about a week after the accident he began to feel very unwell. His skin became hot, he suffered from alternate chills and flushings, with nausea and anorexia. The skin assumed a deep yellow colour, the stools were light and constipated, the urine bile-tinged. This state of things continued for three weeks. He then got well. The second case cited by Mr. Smith is one in which the jaundice followed upon venous,

instead of, as in the above case, arterial, hæmorrhage. The sufferer was a farmer, æt. 56, who lost "an immense quantity of blood" from the giving way of a varicose vein in consequence of an ulcer. After considerable febrile disturbance, the jaundice set in on the fifth day. He recovered in the same manner as the above-mentioned patient did.

These being cases of jaundice following upon hæmorrhage, it may be well for me now to call special attention to the fact that it is much more common for us to meet with hæmorrhage following upon, or at least coincident with, jaundice. From the circumstance that the same condition of liver induces both hæmorrhage and jaundice, namely, hepatic congestion and induration. Notwithstanding my familiarity with all kinds of cases of liver disease, it was long before the vast importance of this fact dawned upon me, or even indeed that I became thoroughly conscious of how exceedingly often the hæmorrhagic diathesis is coincident with, if not even dependent upon, liver derangements. Once the idea had, however, clearly formulated itself in my mind, the pathological haze gradually disappeared, and many cases the *rationale* of which had previously appeared to me perfectly inexplicable became seemingly intelligible.

I shall now relate a case of hæmorrhage presenting us with a rather unusual mode of termination in a person with an enlarged and indurated liver, which, from its having come before me at the time I was giving considerable thought to the probable pathology of such cases, made a deep impression on my mind. There are special reasons for my suppressing alike names of persons and places in this case, so I will merely say that the patient was a powerfully-built, stout, and somewhat eccentric millionaire, living in a large country mansion, within fifty

miles from London. He was unmarried, and aged 36. As he had no great amount of mental resource, lived alone, and possessed an exceptionally fine wine-cellar, drinking, if not eating, was one of his foibles. He was by no means, however, a drunkard, but from living freely, taking little exercise, and being naturally of a somewhat bilious constitution, his liver got enlarged and indurated. It was not, however, on this account that I was asked to see him; but it was from his being suddenly attacked with a violent epistaxis, and his skin being of a yellowish hue, that I was asked to accompany his London consultant to visit him in the country. To make a long story short, the nose-bleeding was so severe and so often repeated (in spite of plugging and perchloride of iron) that I was four times called to his bedside within fourteen days. At length the hæmorrhagic attacks were subdued, and both I and his other two medical attendants thought he would soon be quite convalescent. Within a week, however, his London consultant called upon me with a telegram in his hand, stating that we were wanted immediately, as the patient had been suddenly attacked with acute pneumonia. This astonished us both, as we knew he had not been out of the house, and from our not apprehending the extreme urgency of the call and being full of town engagements, we telegraphed back that we would go by the 3 o'clock train. We accordingly went by it. On our arriving at the country railway station, we were, however, met by a messenger who told us that we need not take the further eight miles drive as the patient was dead. The account I afterwards received was that at about eleven o'clock on the preceding night (the patient would not have a nurse, and was waited upon on alternate nights by his butler, and coachman) the patient had told

the coachman to open the bed-room window in order to air the room. Both patient and attendant, it would seem, almost immediately afterwards, fell asleep, for nothing more is known of what happened, until about five in the morning, when the coachman said he awoke (feeling dreadfully cold, the fire having gone out and the night being frosty) and found his master breathing with difficulty, and complaining of great chest discomfort. His local surgeon was accordingly at once sent for, and on his arrival about eight o'clock he found the gentleman labouring under an attack of acute pneumonia; which by ten o'clock had become so severe that he telegraphed for the usual London consultant to come and see the case. From the pneumonia being of an asthenic character, and the patient's constitution greatly debilitated by the profuse and repeated attacks of epistaxis he had suffered from during the previous fourteen days, the disease ran on with such rapid strides that within twelve hours from the calculated commencement of the attack the man was a corpse. This was a most unexpected ending to a case of nose-bleeding from hepatic disease, within five days from the time we had considered the patient entirely out of danger, and shows how other fatal risks than those arising from the mere direct loss of blood are liable to be encountered in hepatic cases. For here we have an instance of death from asthenic pneumonia, following upon epistaxis, which, in its turn, like the jaundiced complexion of the skin, was the offspring of an enlarged, and indurated liver.

The Nose-Bleedings of Childhood.

Every one is familiar with the nose-bleedings of his school, and college days, and many, I daresay, have, like myself, wondered why in early life, when the coats of the blood-vessels ought to be in a perfectly healthy state, blood should pour forth from them, not alone on the slightest injury, but even without any apparent exciting cause. Sometimes the blood flows from the nose in drops only; sometimes in a full stream; sometimes from one nostril; sometimes from the other; rarely from both at the same time, even though the exciting cause may have been local violence. When the nose-bleeding is purely spontaneous, it recurs at irregular intervals. Sometimes perhaps for two or three days running, then not again for a month or two. As everything in nature has a cause, these periodic nose-bleedings must have one too. What then is it? The martyr to the affection (which, in the majority of instances, is of little consequence, though sometimes it leads to most deleterious results) may present no visible signs of ill-health. On the contrary, he may appear to be of robust constitution, and consequently lead the doctor, as well as the friends, to believe that the bleeding is only a wise provision of Nature to relieve the system of plethora. Can this be so? Surely not. For if Nature be credited with such sagacity, it, at the same time, compels us to regard her as anything but clear-sighted. For, if she possesses the innate wisdom to employ a method of her own to counteract the deleterious plethora, how happens it, one might ask, that she has not the prescience to perceive that, had she refrained from inducing the plethora, she would not be called upon to relieve it? The theory of Nature's

healing sagacity, I fear, must be abandoned, and we must search for another. In cases of nose-bleeding, where there is no visible cause, no ulceration, no polypus, nor any other detectable morbid anatomical lesion in the nostril, and no signs of any renal or heart affections, if the sufferer be neither of a scrofulous, scorbutic, or hæmorrhagic constitution, the cause of the bleeding will probably, I believe, be found to exist in the liver. Liver congestions are particularly common in childhood and early youth. The frequent bilious attacks of boyhood, have this source; and it is easily comprehensible why this should be so, when we recall to mind the much greater functional activity of the liver in infancy than in old age, and that the organ is actually twice as big, in proportion to bodily weight, in childhood as it is in adult life.

Moreover, this theory that many of the cases of nose-bleedings in childhood and early adult life are consequent upon hepatic derangement is strengthened by the fact that in numerous instances, not only have the sufferers a sallow look, and yellow conjunctivæ, but the hæmorrhages at least diminish, if they do not entirely cease, when the liver functions are by judicious treatment set right.

Having treated the subject of hæmorrhages from hepatic causes at such length, I ought perhaps not to omit to say a few words on the umbilical hæmorrhages of the new-born, which most writers assert are almost the invariable accompaniment of infantile jaundice. Some —like Dr. West (p. 622 of his Diseases of Childhood)— even declare the bleeding to be due "to a congenital malformation of the hepatic ducts." I attribute it—as fully explained at p. 304 of my book on the Liver—not to the ducts, but to a congenital malformation in the

blood-vessels and navel, which is, in many cases, no doubt, associated with an equal deficiency in the development of the bile ducts. The two conditions are, however, only associated, not dependent on each other as cause and effect; at least, such is my present opinion.

Treatment of Hæmorrhages from Hepatic Causes.

If many nose-bleedings, hæmatemesis, hæmaturias, menorrhagias, melænas, and even some apoplexies, are not only coincident with, but in reality dependent upon, liver disease, it is evident that their treatment must in some respects differ from similar hæmorrhages with entirely different exciting causes. Of the ordinary forms of treating losses of blood I need not speak, as all are familiar with them; but to the special modes of treating hæmorrhages, the offspring of liver disease, I must direct attention.

As no two constitutions are alike, any more than any two human faces are exactly identical—though there is a general similiarity in all—and every form of disease, even of the same kind, has its own individual characteristics, instead of attempting to indicate particular lines of procedure in individual cases with the hope that they may suit the case, or cases, my readers are likely to encounter, I think the space at my disposal will be more profitably occupied by my laying down some general rules of guidance, which the practitioner may adopt or modify according to his own ideas of the requirements of the case in hand. With this object in view, then, I shall

put forward what I have to say on the *rationale* and treatment of liver-excited hæmorrhages in a categorical series of paragraphs.

To begin with, I shall call attention to the fact that, as hæmorrhages arising from hepatic causes are not unfrequently associated with jaundice, it is well to bear in mind that whenever bile gets into the circulation it renders the blood prone to exude through the coats of the veins, even when no actual lesion exists. This is evidenced by the fact (among others) that sanguineous ascitic fluid is occasionally withdrawn from the abdomen in hepatic cases, where no lesion whatever is detectable in the walls of any of the abdominal blood-vessels after death. Consequently, persons possessing even a slight hæmorrhagic tendency—bleeders, as they are sometimes called—have occasionally profuse losses of blood from merely trivial attacks of liver disease associated with yellow skin. Such, for example, as a transient hepatic congestion.

When liver disease is either the predisposing or the exciting cause of hæmorrhage, the bleeding may occur from more than one orifice of the body at the same time. From mouth and anus, for example, or from nose, urethra, and rectum.

It is not the bleeding in these cases, but the original cause of the bleeding which merits chief attention, and requires immediately to be attacked. For when the cause is remedied the effect ceases.

It must be borne in mind that patients with liver disease often die quite suddenly from hæmorrhages without any blood appearing externally, and that the sudden collapse in such cases is not unlikely to be erroneously attributed to cerebral apoplexy, which it so closely resembles in all respects, except in the absence

of oppressed or stertorous breathing, as to have been repeatedly mistaken for it by men of no mean diagnostic powers. This more particularly occurs when there is profuse as well as sudden hæmorrhage, into the stomach, intestines, or abdominal cavity. And in such cases it is all the more necessary to differentiate this collapse from the coma of cerebral apoplexy, from the treatment suitable to the one, especially that by purgatives, being most pernicious in the other. The administration of a purgative would encourage intestinal hæmorrhage by increasing the peristaltic activity of the bowels; besides which, the timely subcutaneous injection of sulphuric ether, which might restore to consciousness the patient labouring under a temporary collapse induced by a simple loss of blood, would not only be of no avail, but might actually prove detrimental if employed in a case of apoplexy.

It ought equally to be remembered that in the case of persons having a tendency to apoplexy, either from their having atheromatous degeneration of the arterial system, or other predisposing cause, the increased blood-tension induced by the constriction of the vena cava in liver disease (of a hypertrophic or atrophic character) is a formidable exciting cause of cerebral blood-extravasations. Consequently, as a preventative measure, the condition of the liver ought, in persons with an apoplectic tendency, to be carefully attended to.

In all cases of obstruction of the venous system from liver disease, and more especially in those associated with jaundice, the anæmic, equally with the plethoric, are prone to be attacked with hæmorrhages. The only difference demanding attention in the two set of cases being, that while a small loss of blood may be hazardous to the life of an anæmic patient, even a copious bleeding

(especially if there be an apoplectic tendency) may prove advantageous in a plethoric person.

In the latter set of cases, then, Nature's depleting process need not be interfered with until it becomes excessive. By this remark, however, I do not desire it to be imagined that I am a believer in the old pathological dogma of "Nature's therapeutical sagacity," any more than I should wish it to be supposed that I was unemancipated from the equally erroneous old scientific one of "Nature abhorring a vacuum." For when the nose bleeds in an apoplectically-constituted patient it would be unwise to imagine that the hæmorrhage was called forth by beneficent design. To look upon it and treat it as the offspring of Nature's therapeutical sagacity would be a most baneful doctrine for a practitioner to foster. As it might lead him to fold his hands and do nothing while the life of his patient was slipping away. The cause of the blood-vessel in the nose giving way instead of the one in the brain substance, in the case of a person with a predisposition to apoplexy, is simply from the coats of the nasal vessel happening to be the weakest of the two, and the blood bursting forth from the weaker, by diminishing arterial tension, prevents the stronger-coated cerebral vessel giving way and producing an apoplexy. The nose-bleeding is salutary, then, simply by necessity, and not by beneficent design, and we should be guided in the treatment accordingly. It may be laid down as a general rule that it is good policy in the ordinary run of such cases to diminish the arterial blood tension by administering daily a saline purgative.

The object of medicine being to conquer disease and postpone death, from its being always easier to extinguish a spark than a blaze, the first sign of bleeding in all hepatic cases ought to be immediately, when possible,

arrested—no matter whether the bleeding be from mouth or rectum, nose or urethra.

The remedies selected must, of course, vary according to the cause. Hæmorrhage from a perforating gall-stone or hydatid, for example, requires a totally different system of treatment from one caused by a hepatic constriction of the vena cava; for while ice-bags and cold applications to the abdomen, as near as possible to the seat of rupture, are our sheet-anchors in the former, the administration of a smart purgative, either mercurial or saline, is the best mode of treating the latter. Except when the constriction of the vena cava is the result of hepatic atrophy, in which case, as a rule, there is little or no engorgement of the organ to combat; and then we have to fall back on styptics. These may be employed either in the form of hypodermic ergotine injections or of powerful anti-hæmorrhagic mixtures. The one in which I have most faith, and which I have equally employed in nasal, stomachal, renal, and vaginal hæmorrhages from hepatic causes, is the following:—

℞. Pulv. alumnis, gr. x.;
Acidi sulph. aromat., ♏ xxv.;
Ferri sulphatis, gr j.;
Syrupi tolutani, ʒij. M.

In all cases of bleedings induced by liver affections, stimulants, unless to counteract temporary syncope, are to be scrupulously eschewed. Equally so all hot-spiced foods, including peppers and curries in any form. Animal food ought to be given sparingly, but white fish and vegetables freely. Salted meat, salmon, eels, mackerel, lobsters, and crabs, being difficult of digestion, ought to be avoided. Coffee and chocolate, for similar reasons, are not so good as cocoa, milk, and tea. During the con-

tinuance of the hæmorrhage all food is to be taken cold, and all drink iced.

While the bleeding into any portion of the digestive canal is going on the action of the bowels is to be suppressed by the addition of an opiate to the astringent.

In cases of bleedings from piles, or even when the blood exudes from vessels higher up in the rectum, astringent opiate suppositories are exceedingly useful. Hæmorrhage from the urethra is frequently kept under control by the administration of hazeline in two-drachm doses.

In cases of chronic bloody diarrhœa my favourite remedies are a mixture of iron, bismuth liquor, and krameria, and pills consisting of :—

℞ Morphiæ sulphatis, gr. ¼. ;
Ferri sulphatis, gr. j. ;
Pulv. aluminis, gr. iiss ;
Ext. hyoscyami, q. s. fiat pil.

A pill be given as often as is deemed desirable.

As circumstances materially alter cases, the foregoing are to be regarded as mere samples of *forms of remedies*, and nothing more.

I cannot leave the subject of the treatment of liver-induced hæmorrhages without adding a word on the treatment of epistaxis by plugging the nostrils. For although everyone seems to imagine he can do this admirably, it has been my lot, on more than one occasion, to see it, as I thought, bunglingly done. The following simple particulars, I think, ought to be attended to :—

a. The conical plug of surgical lint should be made of not less than two inches in length, its base being of a somewhat greater diameter than the orifice of the nostril to be plugged, and its apex tapered to a flexible point.

b. It should be saturated immediately before insertion

in a solution consisting of one part of the tincture of the perchloride of iron and two parts water.

c. It ought to be introduced into the nostril straight upwards by a firm, but graduated and gentle rotatory motion.

d. Once introduced, it should not be touched for at least twenty-four hours. If the patients complain much of its discomfort, as they often do, soothe it by a narcotic.

e. There is no necessity to plug the posterior nares until it is found that the plugging of the anterior is unsuccessful. For the hæmorrhage is in general from the front part of the nostrils.

f. Before withdrawing a plug one ought always to have another ready at hand, to be immediately inserted should it be found that the hæmorrhage has not ceased.

It is unwise, I believe, to speak of a case of hæmorrhage, even from the nose, if it be the result of hepatic disease, as a trifling matter, either to the friends or the patient. For according to my experience it is much oftener dangerous than most practitioners seem to imagine. One should likewise always bear in mind that the patient is not necessarily safe when the hæmorrhage is arrested. For, as was shown by the fatal case of asthenic pneumonia after epistaxis recorded at p. 113 an unexpected and rapidly fatal issue may even ensue after the patient has been supposed to be entirely out of danger by his medical attendants.

Hepatic Abscesses.

Having, in the first chapter of this series of essays, said that it is mainly due to our not having been clearly taught the signs and symptoms of hepatic suppuration, that we in so many instances fail to recognise them when they are actually before our very eyes. I shall now do my best to try and remedy the defective teachings of our schools, by giving an epitome of what I consider are the most characteristic signs and symptoms of abscess of the liver, and showing how not only palpation and percussion, but the thermometer, and exploring trocar may be made to render most important service in this, which is at best, but an obscure field of diagnosis.

Of the various causes of liver suppurations, I need not further speak. But before adducing the symptoms, I shall embody in the form of a series of aphorisms, a number of clinical and pathological facts connected with hepatic abscesses, which I think it behoves the would-be diagnostician to engrave upon his memory if he desires to facilitate his diagnosing a case of liver abscess.

The three varieties of idiopathic, traumatic, and metastatic abscess of the liver occur in all countries and all climates, in varying proportions according to the relative frequency of their predisposing and exciting causes.

There is no such thing as a spontaneous hepatic abscess. Every suppuration of the liver has, if not a predisposing, at least, an exciting, cause.

The predisposing causes are of two kinds: *a.* An hereditary constitutional tendency to liver disease. *b.* An acquired tendency from an over indulgence in food and drink. The exciting causes are equally of two kinds:

a. One derived directly from without. For example, chills, injuries, and parasites. *b*. One generated within. Suppurations, gall-stones, and embolisms.

Pathologically speaking, all abscesses of the liver naturally divide themselves into two groups:—

a. Those essentially primarily local, including the two forms of idiopathic and traumatic.

b. Those essentially secondary, including the pyæmic and metastatic varieties.

The pathology of all forms of liver abscess is the same, though materially modified by the nature of their exciting causes.

Hepatic abscesses vary in size from that of a walnut to that of the whole liver. Two and a quarter gallons of pus have been evacuated from one. Sometimes the entire liver tissue is broken down and the capsule of Glisson simply forms the sac of the abscess.

Abscesses are far more common in the right, than in the left, lobe of the liver. When multiple they are frequently met with in both lobes.

An abscess may form in the liver at any period of life between early infancy and advanced age.

Suppuration may occur in an atrophied, as well as in a hypertrophied; in a fatty, as well as in a cancerous, liver.

Jaundice is in no case a necessary concomitant of liver abscess. Indeed, it is most frequently absent.

The signs and symptoms are nearly identical in the three varieties. The constitutional peculiarities of the patient alone modifying them.

Hepatic abscess is more common among men than women.

I shall now propound a new, and, I believe, a true idea, regarding the pathology of all liver suppurations.

From the general tenour of this essay it cannot fail to have been observed that I am one of those who view the functions of the human body as the outcome of the same chemico-physical processes that are everywhere at work in the universe at large. Vitality being the climax of co-ordinated chemico-physical action. Health the manifestation of its equipollation, disease the offspring of its perturbation. Hence, I am of opinion that we, as the interpreters of living morbid phenomena, ought to take the same philosophic and wide grasp of natural law while studying the diseased conditions encountered in the human frame, as the pure scientist does in the investigations of the various phenomena met with in the physical world. Were we to reason on this principle in studying disease, I believe, our ideas of the pathology, not only of liver suppurations, but of everything else, would be immensely simplified. I do not see why we should hesitate on religious grounds to think that the Almighty acts in precisely the same way in human beings as he does in the universe at large. Knowing as we do, that he retaineth the big mountain and the little mole-hill in their respective positions on the earth's surface by exactly the same physical agency, why should we imagine it would be derogatory to Him, were we to think that He acts in the animate, as well as in the inanimate, world on one grand harmonious and universally applicable plan, instead of employing a variety of means to produce similar results in different bodies? I for one at least, will believe he does until I have direct evidence to the contrary. Reasoning thus, I am of opinion that the *modus operandi* of every liver suppuration is not only identical—be it caused by the introduction of living and multiplying disease organisms from without, or the generation of inanimate substances, such as

gall-stones, embolisms or fragments of dead liver parenchyma, within—but in no wise differs from that of a suppuration in the finger after the introduction under its skin of a piece of splinter. One and all being but an effort of Nature to expel, after its own manner, an irritative foreign intruder.

Viewed in this light the pathology of hepatic abscesses appears to me to be immensely simplified.

Symptoms Common to all Forms of Liver Suppurations.

Suppuration of the liver is always preceded by a longer or shorter period of hepatitis. The intensity of the pain, however, is in no instance, as I have already shown in the chapter on hepatitis, a criterion of its severity. For we may have great pain with slight inflammation, and scarcely any pain with a considerable suppuration. A superficial abscess is always more painful than a deep-seated one. If, during the ordinary course of a hepatitis the patient should suddenly complain of chilliness or shivering,—when there is no reason to suspect occlusion of the bile duct—and the rigor be immediately followed by an increase of temperature, and an aggravation of the constitutional disturbance, while at the same time the hepatic tenderness is increased, suppuration may be suspected to have set in.

There is always a disinclination to take food, and if the suppuration has existed for some time, there is frequently a well-marked diarrhœa, with or without blood. Vomiting also occasionally occurs; but it is not a common symptom. In some, though not in all, cases of liver abscess,

sneezing, coughing, and deep inspiration causes pain, and then the patient cannot lie comfortably on his side.

The most notable sign of liver abscess, when there exists the before mentioned hepatic symptoms is in my opinion, high temperature, varying from between 102° and 105°. The fever is often associated with hectic and sweating exacerbations. The more superficial the abscess is, the easier is it diagnosed. When situated at the lower margin of the liver, where it projects from under the false ribs the abscess appears as a smooth, hard swelling with a feeling of more or less distinct fluctuation about it. On the other hand when it is situated within the margin of the ribs, palpation furnishes no signs whatever of its existence. Even firm pressure causes no increase of pain, beyond, perhaps, a feeling of a more diffused discomfort. Smart percussion on the other hand in general cannot be readily tolerated. When the abscess is large it causes bulging of the intercostal spaces, even before it actually points, and fluctuation may then be perceptible. But this is not often the case until it points, even when the abscess is below the ribs. To sum up then, it may be said that all that we have to guide us in the diagnosis of the majority of liver abscesses is the existence of pain in the liver, increased on pressure, associated with rigors and high temperature, and the other constitutional symptoms above mentioned. But as the combination of even all of these is not always conclusive, in cases of doubt it is true wisdom to have recourse to an exploring trocar.

The one I recommend to be employed, in the general run of cases, is a fine six inch long French instrument, but one eight inches long, of the diameter of a No. 3 English catheter may be used. There is, comparatively speaking, but little risk in the employment of either, if

care be taken not to insert it too near the ribs, so as to avoid the danger of the instrument being either bent or broken by the ribs during a sudden and forcible act of inspiration. And keeping the point of the instrument from the vicinity of the larger blood-vessels in the neighbourhood of the transverse fissure.

Treatment of Liver Abscess.

As no abscess comes in a moment, and the patient is usually under medical observation some time before actual suppuration of the liver begins, and hepatitis being its invariable precursor, in order to avoid repetition, for its premonitory treatment, I beg to refer the reader back to what I said regarding the treatment of liver inflammations, page 69.

When once a collection of purulent matter has formed in the liver, in so far as the question of treatment is concerned, its exciting cause is of no consequence. For, whether the abscess be idiopathic, traumatic, or metastatic, our sole object is to empty it of its contents, as speedily and as safely as possible. Otherwise the patient may suddenly die, without the slightest warning, from its bursting into the pleura, lungs, pericardium, digestive canal, peritoneum, pelvis of the kidney or even into a blood vessel. Or on the other hand it may kill the patient equally effectually, though more slowly, by hectic, blood-poisoning, or sheer exhaustion. The bursting of an abscess may be said to be its natural mode of termination, and fortunately its thus emptying itself is not always attended with a fatal result. For some patients recover after a liver abscess has evacuated itself either

into the digestive canal, pelvis of the kidney or lungs. Whereas, when the abscess attempts its own cure by any of the other above-named modes it is in general followed by fatal consequences.

The best plan of opening a liver abscess which has not pointed is, I believe, by a trocar, either with or without the aid of an aspirator. Many advise a free incision being made with a knife; but in my opinion when the abscess, has not pointed, such a mode of procedure but adds to the risk, without yielding any proportionate advantage to the patient. In any case, after evacuation of the pus a drainage tube ought to be inserted into the sac of the abscess, and the cavity daily well washed out with tepid antiseptic water. At the same time germicides, in the form of salicylic, carbolic, or mineral acids, along with quinine, should be given, and the general health of the patient carefully attended to. For even in the best of cases a liver abscess is invariably a most exhausting and dangerous affection.

I shall now briefly relate a series of illustrative cases of abscesses occurring in the livers of persons who have never resided in a tropical climate, and as I have only space enough to relate six, I shall give three that proved fatal, and three in which the patients not only recovered, but are still alive. Each case, moreover, I shall select with the view of illustrating some special characteristic, in order that a tolerably good idea of the history, symptoms, and nature of liver abscesses in general may be gleaned from their perusal. I shall begin with the fatal cases.

Predisposing cause—heredity. Exciting cause supposed to have been stimulants. The case is that of a lady professional singer, who I saw along with Dr. Buck. She was a Londoner born and bred, and æt. 57 at the time of her death. For several months previously she was

under treatment for an enlarged, and tender liver. The organ when I first examined it reached to the umbilicus. A couple of weeks later she was attacked with hæmorrhagic diarrhœa—it might be called dysentery—for on one occasion she had twenty-four bloody stools in as many hours. Two months afterwards I punctured the capsule of her distended liver, along with Dr. Buck, in six different places, and it rapidly became reduced in size; but six weeks later it was found (at the post-mortem) to be still more than twice its natural weight, weighing 8lb. 10oz. In the right much enlarged and hardened lobe; towards the convex surface, and near its union with the left lobe, was found an abscess, the size of a bantam's egg. The left lobe of the liver was in an unusually advanced stage of fatty degeneration, the tissue being yellow and oily looking, and a section when examined under the microscope presenting the appearances shown in Fig. 4, which, though in reality a wood-cut of a fattily degenerated liver of a cat, could not possibly represent the hepatic cells loaded with oil granules and globules, and the field filled with oily matters, better had it been drawn from the specimen itself.

FIG. 4.

Fatty degenerated liver, showing the entire loss of the normal granular appearance of the hepatic cells, their place being occupied by oily matters. (See author's Microscopical Demonstrations, Healthy and Morbid, p. 195.)

The right lobe, though also fatty, was in a very much less advanced stage. To the naked eye it simply presented the yellowish grey appearance of an ordinarily slightly fatty organ. Fatty degeneration, as we know, may not only occur in any tissue, but in almost anycondition of tissue. It occurs in a bone as well as in a muscle; in a fibrous tumour as well as in an encephaloid cancer. So there is nothing extraordinary about its having occurred in this case of hypertrophied indurated liver. The case terminated by the patient becoming delirious and dying from exhaustion.

The next case I give as a marked contrast to this, both as regards its history and mode of termination. It is one in which the predisposing cause was over-eating and drinking, and the exciting cause most probably a chill. The case is the one I saw along with Dr. Gandy, already referred to at page 13 as having proved fatal by the bursting of the abscess into the lungs and its contents suddenly suffocating the patient. Its history is briefly this:—The gentleman, æt. 43, a native of the United States of America, up till the 26th year of his age, was a total abstainer, and lived an active, outdoor life. Afterwards becoming very rich, his habits entirely changed. He ceased doing manual work, and lived more than well, for he confessed that he had both eaten and drunk more than was good for him, especially during the last ten years of his life, which he had spent in England. The result being that his liver began to be unable to do its work, and about a year before I saw him, after catching cold, it became congested and tender. Subsequently he had an attack of hepatitis, and at length the organ suppurated. When I first examined his liver it was not only greatly enlarged, but painful on percussion. His abdomen was ascitic, and he had profuse diarrhœa, but no blood in the stools.

His temperature was high, and his skin freely perspiring. It was arranged that I should puncture the organ; but on arriving at his house with the view of performing the operation, he stoutly refused to allow it to be done. Sympathetic pneumonia set in, and, as was shown a few days afterwards by the abscess bursting and emptying itself through the lung, adhesions had taken place; on the one side between liver and diaphragm, on the other between diaphragm and lung. He died suffocated from the hepatic pus filling his bronchi.

The next case is one of metastatic abscess, and is selected not only as showing how a trifling amount of pus in the digestive canal may induce a secondary abscess in the liver, but likewise how difficult is sometimes the diagnosis. The case fell under my notice when house physician in charge of the fever wards of the Edinburgh Royal Infirmary in 1851. It is briefly as follows.—

A well-built lad of 19 years of age was sent into the fever ward under the impression that he was suffering from typhus fever. His pulse was rapid, his skin hot, his tongue foul and tremulous. The liver, though not enlarged, was excessively tender on percussion. There was neither jaundice nor bilious urine, and the stools were of the normal colour. A few days after admission he was seized with pulmonary symptoms, rapidly got into a hectic, and then into a low typhoid state, and died nine days after admission. At the post-mortem an unsuspected abscess, the size of a swan's egg, was found embedded in the right lobe of the liver. Besides which, two smaller ones, the larger of them about the size of a walnut, were found in the middle lobe of the right lung. On examining the intestines a drop or two of pus was found in the peritoneum, on the outside, and at the apex of the appendix vermiformis. Its cause was soon dis-

covered to be the presence of a thick brass pin, about an inch and a quarter in length, with its head, somewhat green and eroded, pointing downwards, and projecting halfway through the caudal extremity of the appendix vermiformis. This is an excellent illustration of how an abscess of the liver may occur in an otherwise healthy constitution, through the medium of metastasis.

I shall now give an illustration in support of my doctrine that suppuration of the liver is probably an effort of Nature to expel from its tissues an intruder, be he small or be he great. The case I submit is one in which the cause of the formation of pus was an impacted gall-stone, which was, I believe, floated out along with the pus when the abscess burst, after the patient had been for several days at the gates of death. The case is that of a lady, æt. 43, who is still under observation. I first saw her in 1882 along with Mr. J. Burton, of Blackheath; she was then suffering from impacted gall-stone, and was not only jaundiced and had the urine loaded with bile, but not a trace of biliary pigment was visible in the fæces. The first stool I saw was nearly as white as snow. On another occasion the motions had a delicate pinkish mauve-white colour. The case went on very much as most bad ones generally do, until April 1883, when I was called to see her on account of her symptoms having assumed an aggravated form. The above mentioned conditions continued, but superadded to them were signs of abscess. Her pulse was 106, her respirations 34, and her temperature ranging from 100° to 103°. A week later, as Mr. Burton considered her in a critical state I was again called to her bedside. The pulse was 120, the respiration 42, and the temperature at the time of my visit, 5 p.m., 104°, at

11 a.m. it had been 101°. The hepatic tenderness was greatly increased, and there was a continuation of the rigors. But little importance could be attached to their presence, however, in this case, from the probability of their being due more to the impaction of the stone than to the formation of pus. Three days later I again saw her, she had had an attack of epistaxis, and her temperature had gone up another degree. Her pulse was now very feeble, as well as quick, and she was in such a prostrate state that both Mr. Burton and I came to the conclusion that if the abscess did not burst soon, she would assuredly sink and die. Apparently, it did burst. For suddenly her temperature fell to 103°, and soon after to 97°, that is to say to 1 and 4-10ths of a degree below the normal. The liver dulness had also changed from being above, to below normal, reaching indeed only to $2\frac{1}{2}$ inches in the nipple line. From now she began to improve. The patient afterwards told me that although she found no stone, she noticed that her stools on several occasions contained "yellow matter." This is what Mr. Burton said of her in a letter three months later: "I cannot say Mrs. —— is quite well but she is better, and away at the sea-side. When she left, although better in colour, and free from pain, the motions were still without bile." Nothing more need be added to this report, except that I still occasionally see this patient on account of some more gall-stones remaining in her gall-bladder. In other respects she seems to be perfectly well.

Latent liver abscess from direct injury. A gentleman, æt. 35, eighteen months before I saw him, had a fall from a bicycle, the handle of which struck him so violently in the right hypochondrium as to render him

unconscious for a minute or two. He had recovered sufficiently in a fortnight to be able to leave the house, though not free from pain, and walking with difficulty. When he came to me he complained of pain in the liver, under the ribs, and there was a distinct fulness and tenderness on pressure from the lower margin of the liver half way down to the umbilicus. No fluctuation was perceptible. The bowels were very constipated. At the stage the disease was then in the case was a puzzling one to diagnose, but thinking from its history that it might possibly be one of latent hepatic abscess, poultices and hot turpentine cloths were ordered to be applied over the painful parts, and a saline purgative given. Within a fortnight he was attacked with vomiting, and an increase of pain, both of which shortly afterwards suddenly ceased, and very soon afterwards a quantity of a brown pus-like liquid, not a chocolate-coloured one, as is usual in such cases, came away by stool. The swelling was now noticed to have considerably diminished. A week later a second discharge of an exactly similar liquid came away, and immediately afterwards all traces of the swelling disappeared. From that time the patient got rapidly well.

The last case I will direct attention to, is one of a suppurating hepatic hydatid, which nearly killed the patient by blood-poisoning, from the absorption into his system of its obnoxious putrefying pus.

The case has several important features, not the least of which is that its anomalous symptoms led to most contradictory diagnoses by four or five different practitioners one, if not two, thinking it was a case of gall-stone, the others diagnosing it as one of incurable cancer. Its history is briefly this. In the end of 1882 I was asked by Mr. Withers to see a patient in Shrewsbury, where I

met with him in consultation Dr. White and Mr. Brookes. The case was that of a gentleman, æt. 60, who had suffered for some months from enlarged liver, with sudden acute pain and rigors, like those caused by gall-stones. After a time there was found to be a distinct swelling at the lower edge of the liver, and gradually the patient's complexion assumed a cachectic appearance. His complexion appeared to me to be like that of a person labouring under blood-poisoning. What had puzzled his medical attendants was that while the pain and rigors pointed to gall-stones, all the other signs and symptoms of the case accorded most with cancer. My examination of the liver led me to take a different view of the case from the others. I considered that neither gall-stones nor cancer had anything to do with it, and that the swelling was due either to a hydatid or an abscess, and proposed putting this diagnosis to the test of exploration. An exploration was accordingly made, and a quantity of most unusual-smelling pus flowed out. As Mr. Withers said, he had never smelt "such stinking stuff," and most assuredly I never had, for it smelt exactly like what I could fancy putrid cod-liver oil would smell. An aspirator was next sent for, and the abscess emptied by Mr Withers in my presence.

During the process of evacuation the end of the canula became repeatedly blocked by hydatid vesicles (?) The case was, in fact, one of suppurating hydatid. Its further history I take from Mr. Withers, Mr. Brookes, and the patient's subsequent correspondence. "The patient had no febrile disturbance after the operation. He slept well; but on the following day the tumour was rather larger and more tender than before." Then for a day or two "it seemed to diminish." While again another change took place. "It became prominent with

distinct fluctuation and six or eight drachms of very fœtid pus was drawn off, and a drainage tube introduced, after washing out the cavity with a carbolic solution." On the thirty-eighth day after the tumour was explored, and emptied, I received a letter saying:— "The case is still going on well, but there is still a copious discharge. The sac is contracting." Soon afterwards the patient was up and about. Three years have elapsed since then, and within the last few days I have received from the patient a letter which ends with these words, "I have enjoyed uninterrupted good health up to the present time."

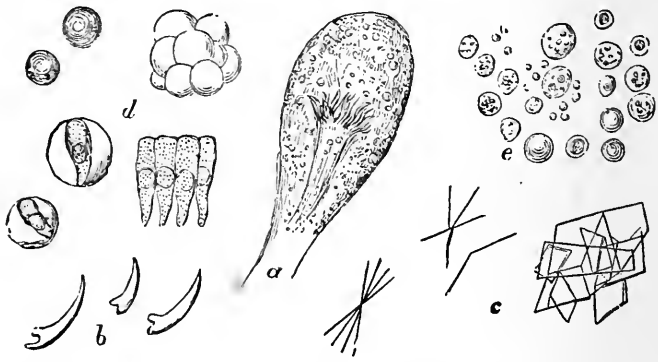
The result of this case appears to support the general idea that correct diagnosis is superior to wise treatment, as without the former it is impossible to have the latter, and all know that when the diagnosis is correct, the treatment we adopt is seldom wrong. The probability is that had this hydatid not been explored and emptied of its contents, the patient would have succumbed to the effects of blood-poisoning from the continuous absorption into his system of the putrid purulent matter.

As in cases of the above kind it occasionally happens that there exists a doubt whether the withdrawn pus be the contents of a suppurating hydatid cyst, or of a true abscess, it is well to examine it with the microscope. For should it be the former, in addition to pus corpuscles, one in general encounters some of the elements of the broken up entozoon. Unless it be in an advanced stage of putrescence. The subjoined woodcut shows what one may expect to meet with in a case of suppurating hydatid.

It is a copy of one kindly lent me by Mr. Jabez Hogg to illustrate the abstract I gave (at p. 960 of my book on the Liver) of his interesting case of degenerating hepatic

hydatid, published in the Pathological Society's Transactions of 1857 (p. 545). I know of no better illustration of the microscopical elements met with in such cases than this of Mr. Hogg's case.

Fig. 5.



a. Cyst with echinoccocus within it. *b*. Hooklets of the entozoon. *c*. Crystals of cholesterin. *d*. Cylindrical epithelium. *e*. Pus corpuscles. Mag. 200 diameters.

Those of my readers who may desire to have still fuller information on the subject of hepatic abscess, more especially of the modern ideas of their etiology and treatment I would beg to refer to my book on the Liver. Where they will not only find a *résumé* of them, but the whole subject of liver suppurations treated from some entirely different points of view, from what it has been done here, coupled with an important series of illustrative cases, carefully selected from the most reliable sources.

INDEX.

Abscess of the liver, 15, 51, 125
„ bursting of, 13, 130, 135
„ etiology, 7, 33, 51, 55, 62, 125
„ difficulties in the way of diagnosis, 10, 13, 125, 134, 137
„ frequency of in England, 10, 63
„ latent, and other kinds of, 134
„ mistaken for other diseases, 13, 134, 137
„ symptoms, 128
„ tapping, 97, 130
„ treatment, 130
Apoplexies in liver diseases, 107, 108, 119
Ascites, 90
„ amount of fluid secreted in, 91, 107, 111
„ differential diagnosis of, 93, 119
„ etiology, 90
„ treatment, 94
„ tapping, mode of procedure, 97
Atrophy of liver, 80, 92
„ symptoms, 85
„ treatment, 88
„ varieties, 82
„ leucin and tyrosin in, 87
Biliary concretions, 6, 9, 10, 105, 111, 133, 136
Bleeding the liver, 72
Cancer of liver, 7, 137
Chills a cause of hepatitis and hepatic abscess, 27, 133
Cirrhosis, 80
Climate, effects of, in hepatitis, 33
Diagnosis, 5, 11
„ of abscess, 124
„ of ascites, 90
„ of atrophy of liver, 80, 92
„ of enlarged liver, 64
Diuretics, 94
Dropsy, 90
Drunkard's liver, 18, 36, 80
Dysentery, 25, 63, 131
Embolism a cause of abscess of the liver, 26
Enlargement of the liver, 64
Epistaxis, 107, 111, 116, 135

Exploring the liver, 68, 129, 138
Extravasation of blood in the liver, 109
Fæces in liver disease, 85, 93, 106, 111, 135
Flatulency in liver disease, 86, 89, 93
Food, 88, 121
Gall-stones, 6, 9, 10, 85, 105, 111, 134, 137
„ a cause of abscess of liver, 125, 134
Germs, a cause of liver disease, 56, 60
Gluttony, a cause of liver disease, 18, 125, 133
Hæmaturia, 68, 111
„ paroxysmal, 68
Hæmorrhages in liver diseases, 86, 105, 116
„ in atrophy of the liver, 105, 110
„ in hypertrophy, 105, 107
„ from bladder, 107
„ from bowels, 107, 130
„ from mouth, 106, 112
„ from nose, 107, 111, 113, 116, 135
Hepatic phlebotomy, 72
Hepatitis, 60, 64
„ causes of, 55, 60, 62
„ malarial, 67
„ peri-hepatitis, 66
„ sympathetic, 66
„ syphilitic, 68
„ treatment, 69
„ a cause of hepatic abscess, 55
Hobnail liver, 80, 110
Hydatids, cause of hæmorrhage, 106
Hydatids, cause of abscess, 137
Hypertrophy of liver, 64
Intemperance a cause of liver disease, 18, 36, 80, 133
Jaundice, 66, 85, 112, 126
Leucin, 87
Melæna, 7, 106
Nodulated liver, 82, 110
Nose-bleeding in early life, 107, 116
Operations on liver, 68, 70, 72, 136
Peritonitis in ascites, 100, 102
Pins a cause of hepatic abscess, 133
Puncturing capsule of Glisson, 69, 70, 72, 131
„ abdominal parietes, 97
Pus, gonorrhœal, cause of abscess of liver, 63
„ intestinal, cause of abscess of liver, 63, 133
Rectum, stricture of, 63
Renal ascites, 93
Serum, ascitic fluid, 91
„ secretion of, 91
Statistics of abscesses, 63
Suppurations of liver, 15, 125
„ hydatid, 137,
Syphilitic disease, 68
Tapping in abscess, 137
„ ascites, 97
Treatment of abscess, 130
„ ascites, 94
„ atrophy of liver, 88
„ cirrhosis, 88
Trocars to be used in tapping, 97
Tumour impeding the operation of tapping, 100
Tyrosin in the urine, 87
Urine, 66, 87, 96

T. W. DANKS & CO., PRINTERS, DEAN STREET, SOHO SQUARE, W.